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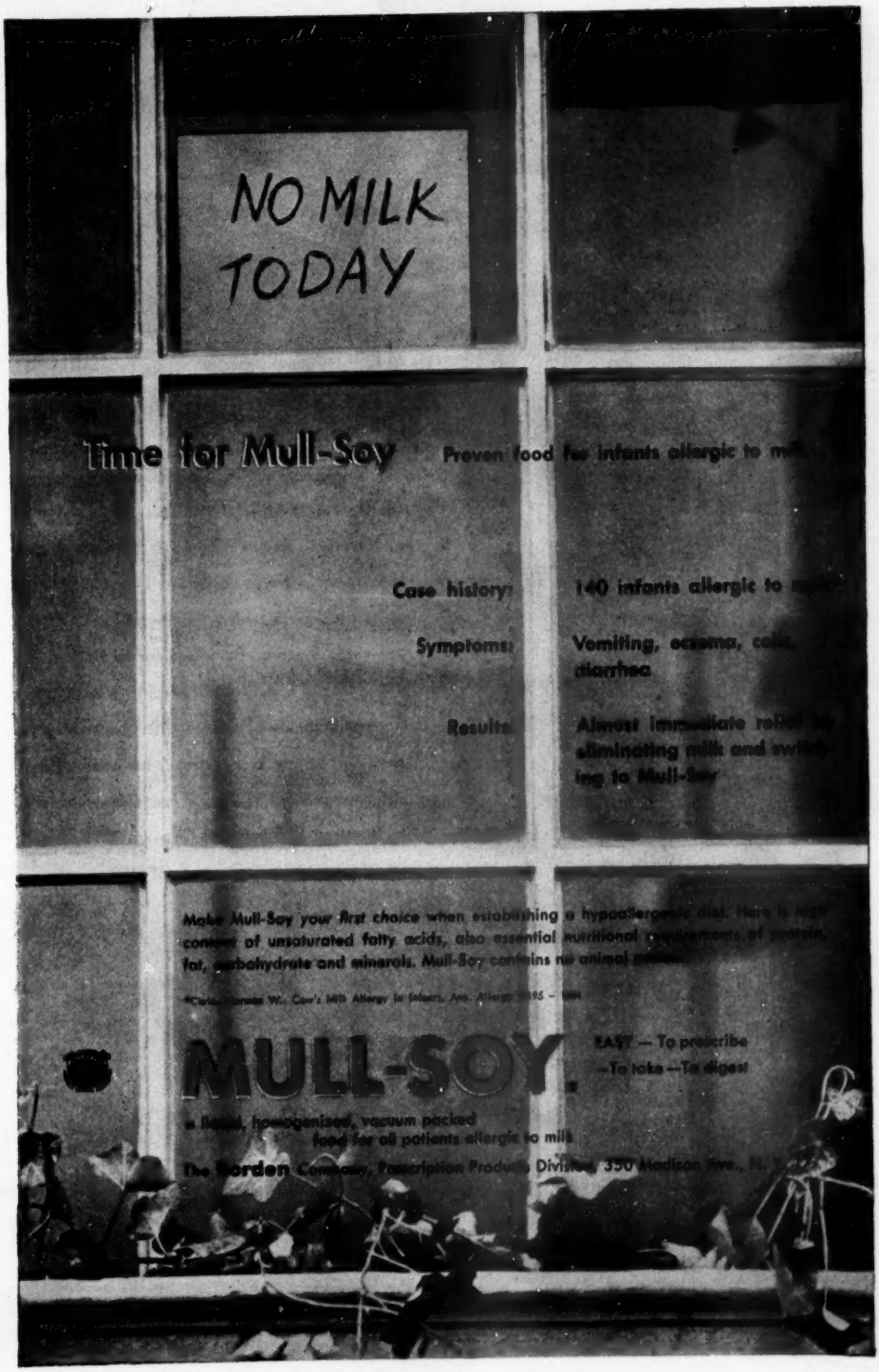
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1. Tisdall, F. F., and Jolliffe, N., in Clinical Nutrition, New York, P. B. Hoeber, 1950, c. 23, p. 590. 2. Sealock, R. R., and Goodland, R. L.: Science *114:* 645 (Dec. 14) 1951.

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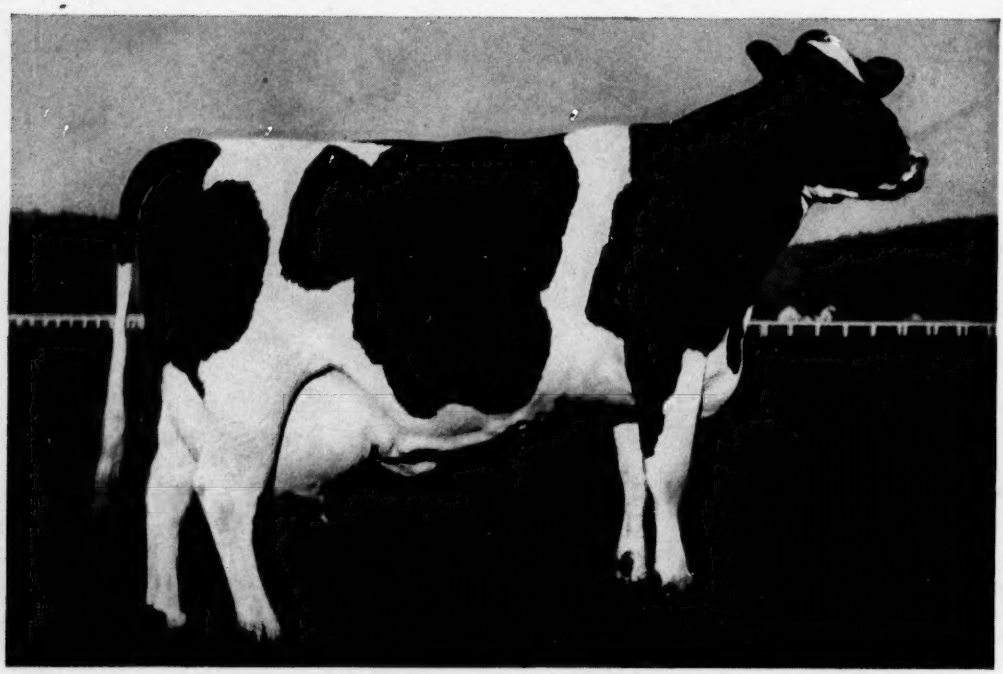

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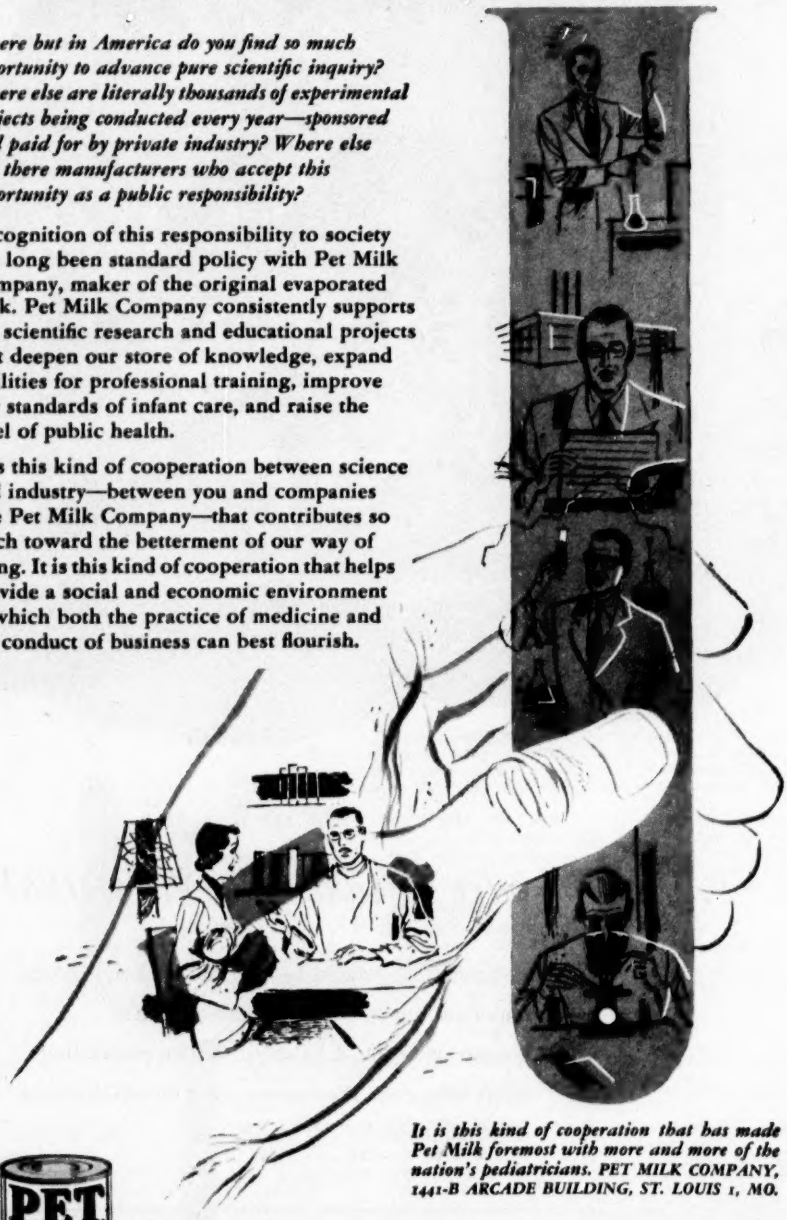

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THYROTROPIC HORMONE IN INFANTS AND CHILDREN

Differentiation Between Primary and Hypopituitary Hypothyroidism

DONALD E. PICKERING, M.D.
AND
EARL R. MILLER, M.D.
SAN FRANCISCO

THIS PAPER presents data on the effect of the thyrotropic hormone [1] on I^{131} uptake by the thyroid for the differentiation of primary from secondary hypothyroidism in infancy and childhood.

While hypothyroidism in infancy and childhood has been the subject of much clinical research since the turn of the century, only minor amendments have been made to the excellent clinical classification presented by Pineles in 1902.[2] Much has been learned, however, about the chemistry and physiology of pituitary and thyroid function, and from these certain practical laboratory aids in diagnosis have been standardized and are available to most clinicians for routine use. Among these, in hypothyroidism, are roentgenographic evidence of retardation of skeletal maturation, failure of linear bone growth, epiphyseal dysgenesis,[3] chemical and metabolic evidence of lowered basal metabolic rate,[4] low serum alkaline phosphatase,[5] elevated serum cholesterol,[4] and hypercarotinemia.[6]

During the past decade the practical and accurate determination of the serum protein-bound iodine concentration has added a comparatively direct method of

From the Departments of Pediatrics and Radiology, University of California Medical School (Dr. Miller).

Supported in part by a research grant from the Playtex Park Research Institute and by contract AT-11-1-GEN-10, Project No. 2, of the University of California with the United States Atomic Energy Commission and in part by the University of California.

1. Thyrotropic hormone, a lyophilized, pyrogen-free product, was furnished through the courtesy of the Armour Laboratories, Chicago. In no case was either a local or a generalized reaction encountered following its dilution with sterile isotonic sodium chloride solution and intramuscular injection.

2. Pineles, F.: Über Thyroplasie (kongenitales Myxödem) und infantiles Myxödem, Wien. klin. Wchnschr. **16**:1129, 1902.

3. Reilly, W. A., and Smyth, F. S.: Stippled Epiphyses with Congenital Hypothyroidism (Cretinoid Epiphyseal Dysgenesis), Am. J. Roentgenol. **40**:675, 1938. Wilkins, L.: Epiphysial Dysgenesis Associated with Hypothyroidism, Am. J. Dis. Child. **61**:13, 1941.

4. Wilkins, L.; Fleischmann, W., and Block, W.: Hypothyroidism in Childhood: The Basal Metabolic Rate, Serum Cholesterol and Urinary Creatine Before Treatment, J. Clin. Endocrinol. **1**:3, 1941.

5. Talbot, N. B.; Hoeffel, G.; Schwachman, H., and Tuohy, E. L.: Serum Phosphatase as an Aid in the Diagnosis of Cretinism and Juvenile Hypothyroidism, Am. J. Dis. Child. **62**:273, 1941.

6. Escamilla, R. F.: Carotinemia in Myxedema: Explanation of Typical Slightly Icteric Tint, J. Clin. Endocrinol. **2**:33, 1942.

measurement of one aspect of thyroid function.[7] With the development of isotopic techniques, I^{131} uptake and excretion studies have similarly added much to our present concepts of thyroid function and have been used in many centers as an aid in diagnosis and treatment of thyroid disorders.[8]

While the above diagnostic methods have aided in the clinician's identification of hypothyroidism per se, they generally have failed to differentiate between hypothyroidism due to thyroid failure, or athyrosis (primary hypothyroidism), and hypothyroidism due to deficiency of anterior pituitary thyrotropic hormone production (secondary hypothyroidism). In general, it has been customary to attempt to make this differential diagnosis through a search for signs of other pituitary deficiencies by a study of other trophic hormones, such as the gonadotropins and corticotropin (ACTH), and of growth. If other pituitary deficiencies are present, it is taken as evidence of pituitary failure. Errors may arise from such parallelism, however, since such deficiencies may be caused by myxedema.[9] This was found to be true in one of the cases to be reported (A. P.). In a hypothyroid patient growth failure out of proportion to clinical and chemical indications of hypothyroidism and poor growth following thyroid therapy suggest the presence of hypopituitarism.

An attempt to differentiate between primary and secondary hypothyroidism reported here is based upon the pronounced effect of parenterally injected thyrotropic hormone on I^{131} uptake in thyroids of patients with pituitary deficiency, in contrast to the slighter effect on those with adequate pituitary function.

EXPERIMENTAL PROCEDURES

Description of Patients.—Six clinically hypothyroid children without goiter were selected for study from the patients attending the University of California Pediatric Clinic in San Francisco. Five clinically euthyroid children were chosen for control. Among the controls one patient had diabetes (P. B.), one was a dwarf (L. T.), two were mentally defective (S. H. and R. E.), and one had Legg-Calvé-Perthes disease (P. F.). The dwarf was normal in all respects, except for stature. The results of studies of his intelligence quotient, body proportions, dental and bone age, gastrointestinal tract, and genitourinary and cardiovascular systems were all within normal limits. Among the hypothyroid patients two were presumed to be athyrotic cretins (R. S. and R. M.); one (A. P., 9 years of age) became myxedematous spontaneously at 5 years of age and remained untreated for four years before investigation. Of the patients with pituitary hypothyroidism studied, one was a dwarfed infant (T. W.) and the other a 15-year-old girl with an eight-month history suggesting anorexia nervosa (B. P.).

7. Riggs, D. S.; Gildea, E. F.; Man, E. B., and Peters, J.: Blood Iodine in Patients with Thyroid Disease, J. Clin. Invest. **20**:345, 1941. Rapport, R. L., and Curtis, G. M.: The Clinical Significance of the Blood, J. Clin. Endocrinol. **10**:735, 1950. Mann, E.; Pickering, D. E.; Walker, J., and Cooke, R. E.: Butanol-Extractable Iodine in the Serum of Infants, Pediatrics **9**:32, 1952.

8. (*a*) Miller, E. R.; Dailey, M. E.; Holmes, A. V.; Alexander, G. L., and Sheline, G.: Studies with Radioiodine: I. Function and Rate of I^{131} Uptake of Thyroid, Radiology **57**:37, 1951. (*b*) Miller, E. R.; Dailey, M. E.; Soley, M. H.; Foreman, N.; Holmes, A. V.; Alexander, G. L., and Sheline, G. E.: Studies with Radioiodine: II. Treatment of Patients with Hyperthyroidism by I^{131}, ibid. **57**:227, 1951. (*c*) Miller, E. R., and Sheline, G. E.: Studies with Radioiodine: III. Problem of Dosage in the Treatment of Hyperthyroidism, ibid. **7**:722, 1951. (*d*) Hamilton, J. G.; Soley, M. H.; Reilly, W. A., and Eichorn, K. B.: Radioactive Iodine Studies in Childhood Hypothyroidism, Am. J. Dis. Child. **66**:495, 1943.

9. Statland, H., and Lerman, J.: Function of Adrenal Cortex in Myxedema, with Some Observations on Pituitary Function, J. Clin. Endocrinol. **10**:1401, 1950.

Studies of Patients.—All the patients were studied clinically, and determinations of bone age and protein-bound iodine were made for each. Pituitary-adrenal function studies were made for three of the controls and all of the hypothyroid patients (Table 1).[10] At least two of the pituitary-adrenal function tests were carried out on patients designated "normal" in Table 1. Three of the hypothyroid patients had normal results of pituitary-adrenal function studies, and three had results in the abnormal range as follows:

PATIENT A. P.—(1) The 24-hour urinary 17-ketosteroid excretion was low: 1.1 mg. (normal range 2 to 5 mg. per 24 hr.); (2) circulating eosinophiles did not drop significantly (156 to 147 per cubic mm.) four hours after administration of epinephrine or four hours after administration of corticotropin (234 to 184 per cubic mm.); (3) there was no significant increase in urinary 17-ketosteroid excretion after 48 hours of administration of corticotropin.

It is interesting to note that the above pituitary-adrenal test pattern reverted to normal on retesting, after the patient received a three-month period of thyroid medication followed by 30 days without thyroid. This suggests that her originally subnormal adrenal cortical function was secondary to myxedema and that the adrenals regained competency following relief of her myxedema.

TABLE 1.—*Collateral Data on Patients Receiving Thyrotropic Hormone*

Diagnosis	Patient	Age		Bone Age	Basal Metabolic Rate	Pituitary-Adrenal Function	Urinary Gonadotropin, Mouse Units	Protein-Bound Iodine (Serum) γ/100 Cc.
		Chronological, Yr.	Onset of Symptoms					
Euthyroid	L. T.	1	Dwarf from 3 mo.	Normal	..	.....	...	4.7
	P. F.	4	.....	Normal	..	Normal	...	5.5
	S. H.	1½	.....	Normal	..	.....	...	4.7
	P. B.	10	.....	Normal	..	Normal	...	5.0
	R. E.	1½	.....	Normal	..	Normal	...	5.0
Hypothyroid	L. B.	1¾	4 mo.	9 mo.	..	Normal	...	3.8
	R. M.	1½	1½ mo.	2 mo.	..	Normal	...	3.0
	R. S.	5/12	1½ mo.	0	..	Normal	...	2.9
	A. P.	9	6 yr.	6 yr.	—25%	Abnormal	<5	3.1
	T. W.	1	3 mo.	3 mo.	..	Abnormal	<5	3.4
	B. P.[10]	15	14 yr.	Normal	—43%	Abnormal	<5	3.8

PATIENT T. W.—This dwarfed and fragile one-year-old infant demonstrated less than 50% decrease in circulating eosinophiles four hours after administration of epinephrine, while she demonstrated a significant (50% decrease) response to corticotropin. After thyroid therapy she demonstrated much less growth and osseous maturation than would be customarily found in a cretin infant. These findings support the impression of hypopituitarism.

PATIENT B. P.—This 15-year-old girl was well until eight months before investigation, at which time amenorrhea and anorexia associated with marked weight loss developed and her condition was diagnosed as anorexia nervosa. Neurological investigation at the time of our study revealed no evidence suggesting disease of the central nervous system. She demonstrated an adequate response to both epinephrine and corticotropin by more than a 50% reduction in circulating eosinophiles. Twenty-four hour urinary 17-ketosteroid excretion was 2.4 mg., which

10. When there was any question relating to the integrity of pituitary-adrenal function, the following determinations were made: (1) the determination of 24-hour urinary excretion of 17-ketosteroids and Compound F (17-hydroxycorticosterone); (2) the response of circulating eosinophiles to epinephrine in the following doses: 0.03 cc. 1:1,000 epinephrine per kilogram until 0.5 cc.; 0.5 cc. for patients weighing 15 to 30 kg.; 0.75 cc. for larger patients; (3) the response of circulating eosinophiles to parenterally injected corticotropin (ACTH), and (4) the 48-hour response to corticotropin injection as measured by 17-ketosteroid and Compound F excretion in the urine.

is lower than normal for a girl in this age group. Forty-eight hours after corticotropin therapy her urinary 17-ketosteroid excretion rose precipitously and subsequently returned to her originally low value within 24 hours. Compound F (17-hydroxycorticosterone) excretion followed a similar pattern.

I^{131} AND THYROTROPIC HORMONE STUDIES

Following an 8- to 12-hour fast, each child received from 15 to 40 μc of carrier-free[11] I^{131} orally or through a polyethylene tube. The per cent of uptake of the I^{131} in the thyroid was determined at 0, 2, 6, and 24 hours following administration. The techniques used in handling and counting I^{131} were those described in a previous report from this laboratory, with but slight modification.[6a] The initial 24-hour I^{131} uptake by the control group was from 12 to 20%. The initial 24-hour I^{131} uptake by the thyroid both in cases of hypopituitary hypothyroidism (T. W. and B. P.) and in one case of primary hypothyroidism (L. B.) fell within the range of the euthyroid group (12 to 20%). The uptake of I^{131} in the remaining three hypothyroid patients was very small. I^{131} uptake by the thyroid in hypothyroidism, primary or secondary, may therefore be indistinguishable from that in euthyroid children. Significant I^{131} uptake in cretins has previously been reported by this and other clinics.[12]

TABLE 2.—*Thyrotropic Hormone Dosage (Given Intramuscularly Every 12 Hours × 4)*

Patient's Weight, Kg.	Thyrotropic Hormone, Mg.
5	5
5–15	7.5
15–20	10
20–+	15

Twenty-four hours following completion of the initial I^{131} uptake studies, thyrotropic hormone therapy was begun and was continued over a period of 48 hours for all patients whose cases are reported. This was followed by a repetition of the I^{131} uptake study, employing the same dosage and technique used in the initial study. The quantity of thyrotropic hormone injected per dose is shown in Table 2.

RESULTS AND COMMENT

Data showing the 24-hour thyroid uptake of I^{131} before and after 48 hours of thyrotropic hormone administration are presented in the graph.

Pretreatment per cent uptake (24 hours) of I^{131} by the thyroid is represented in vertical clear columns, below zero in each case. Per cent uptake (24 hours) of I^{131} by the thyroid after 48 hours of thyrotropic hormone therapy is graphed as the entire vertical column, and the difference between the two, per cent increase following administration of thyrotropic hormone, is graphed above zero in the stippled portions of the columns. Individual cases are graphed according to clinical impression in the bottom horizontal column.

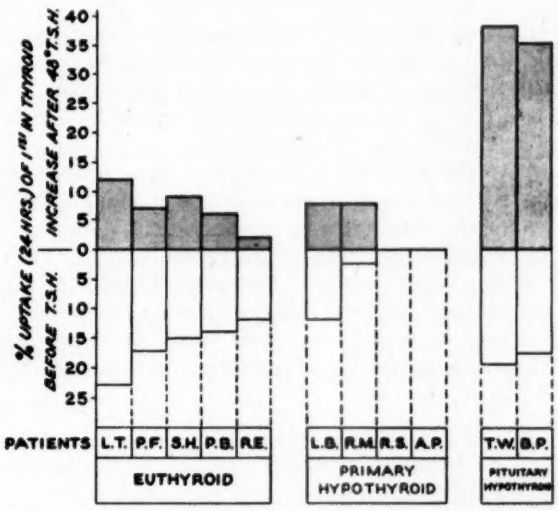
In these euthyroid patients the 24-hour thyroid uptake of I^{131} increased from 2 to 12% after administration of thyrotropic hormone.

11. Radioactive I^{131}, free from nonradioactive iodine, permits labeling of the extra thyroidal inorganic iodine pool without contributing to it.

12. Lerman, J.; Jones, H. W., and Calkins, E.: Studies on 2 Sporadic Cretinous Brothers with Goiter, Together with Some Remarks on the Relation of Hyperplasia to Neoplasia, Ann. Int. Med. **25**:677, 1946. Stanbury, J. B., and Hedge, A. N.: A Study of a Family of Goitrous Cretins, J. Clin. Endocrinol. **10**:1471, 1950. Footnote 8*d*.

The data from studies of the hypothyroid patients divide themselves into two groups. In the first, primary hypothyroidism, the response to the administration of thyrotropic hormone is in the normal range (2 to 12%); in the second group, secondary hypothyroidism, the response is over three times as high (35 to 38%).

One patient with primary hypothyroidism (L. B.) was not recognized to be hypothyroid until she demonstrated a dramatic response to thyroid therapy. Her protein-bound iodine determination was 3.8 γ per 100 cc. An increase of 8% was observed in her 24-hour I^{131} uptake after administration of thyrotropic hormone. Another patient with primary hypothyroidism (R. M.) was an 18-month-old cretin whose clinical diagnosis was made at the age of 2 months. He had been inadequately treated from the time of diagnosis until three weeks before this study was begun, at which time therapy was discontinued. There was no significant uptake



Thyrotropic hormone effect on uptake (24 hour) of I^{131} in thyroid.

of I^{131} by the thyroids in the remaining children with primary hypothyroidism (A. P. and R. S.).

The two hypopituitary children with secondary hypothyroidism (T. W. and B. P.) demonstrated an increase of over 35% in their uptake of I^{131} after administration of thyrotropic hormone. Similar results have been reported in the cases of three adults by Querido and Stanbury.[13] Thus, the use of I^{131} uptake as influenced by the administration of thyrotropic hormone is a practical diagnostic procedure which served to differentiate primary from secondary hypothyroidism in these patients. In accord with previous observations, we have found that the thyroid uptake of I^{131} in primary hypothyroidism may fall within the range for euthyroid patients.

13. Querido, A., and Stanbury, N.: The Response of the Thyroid Gland to Thyrotropic Hormone as an Aid in the Differential Diagnosis of Primary and Secondary Hypothyroidism, J. Clin. Endocrinol. **10**:1192, 1950.

It would seem to us that carrier-free I^{131} uptake represents thyroid uptake of the extrathyroidal inorganic iodine pool. Such uptake need not be directly related to production and/or release of thyroid hormone. It is obviously influenced by the dimensions and concentration of the immediately available 24-hour iodine pool. Thyroid size and exposure to the pool are also determinants. It is also influenced directly by the thyrotropic hormone.

While the per cent of the iodine pool taken up by the thyroid in the hypopituitary-hypothyroid group during the pretreatment period was found comparable to the per cent uptake in the euthyroid group, following thyrotropic hormone administration similar comparison revealed greatly increased uptakes in the hypopituitary hypothyroid group. Such observations suggest existence of a small iodine pool in the hypopituitary hypothyroid.

SUMMARY AND CONCLUSIONS

The effect of thyrotropic hormone as measured by an increase in I^{131} uptake has been measured in five euthyroid and six hypothyroid children.

Parenteral administration of thyrotropic hormone for 48 hours to five euthyroid children was followed by an increase in thyroid I^{131} uptake by 3 to 12% over their preceding 24-hour thyroid uptake of I^{131}.

Two of four hypothyroid children, assumed to have primary end-organ deficiency, demonstrated no I^{131} uptake either before or after thyrotropic hormone administration, while the remaining two demonstrated an increase of 8% of I^{131} uptake following thyrotropic hormone administration.

In contrast, similar administration of thyrotropic hormone in two hypothyroid children who demonstrated collateral signs of pituitary insufficiency was followed by an increase of from 35 to 38% over their previously determined thyroid I^{131} uptake. This suggests a method for differentiating primary from secondary hypothyroidism in childhood.

TRIGGER THUMB IN INFANTS

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AND
WILLIAM E. JENSEN, M.D.
HONOLULU, T. H.

A TRIGGER mechanism is one in which motion is carried to a certain point, at which a resistance is met, but further motion carries it beyond the resistance with a snapping sensation or a definite "give." In the trigger thumb the motion from flexion to extension is most frequently blocked at about 150 degrees, and then further pressure overcomes the resistance and full extension is obtained. This works in a similar way with motion from full extension to flexion, meeting the same resistance. When the child is seen by the physician, the thumb is frequently locked with the distal phalanx in flexion. However, the history given by the parents shows that the typical trigger mechanism was present at the onset, but gradually the resistance increased until the phalanx locked in flexion. In our series of cases seven of the nine trigger thumbs were in the persistent locked state when the condition was observed. Finding the thumb in this state would give reason for an error in diagnosis. During the time these cases of trigger thumbs were collected and observed the same mechanism was not seen in any other digits in children coming to the Shriners' Hospital for Crippled Children.

This condition has also been called snapping thumb and stenosing tenovaginitis. It does not appear progressive after the persistent locking occurs, and except in the report by Rose,[1] writers do not describe any inflammatory changes, but only hyperplastic changes. An example of the stable nature of the condition is the case reported by Compere [2] of a 24-year-old woman who had had the condition since she first noticed it at 4 years of age. In this particular case there had been no evidence of progressive changes. This is especially true because of the fact that there were no progressive degenerative changes that could not be relieved by simple incision of the tendon sheath, which is exactly the same as in infants.

Numerous different diagnoses are made when the true nature of the condition is not recognized. The commonest ones are congenital contracture of the thumb, chronic dislocation of the interphalangeal joint, and ankylosis of the interphalangeal joint. The lack of reports of many series of cases would suggest that this is not a frequently diagnosed condition, or, as considered by some writers, is somewhat rare. The adult type of trigger finger is fairly frequent, but the involvement of the thumb in adults is considered rare. The case reported by Compere [2] is the only one

From the Shriners' Hospital for Crippled Children.

1. Rose, T. F.: Bilateral Trigger Thumb in Infants, M. J. Australia **1**:18 (Jan. 5) 1946.
2. Compere, F. L.: Bilateral Snapping Thumbs, Ann. Surg. **97**:773 (May) 1933.

described that was of the same nature as those found in infants. He reviewed the literature and described reports of various authors of a trigger mechanism involving the tendon of the flexor pollicis longus muscle in adults, but these were of a few months' or years' duration and not related definitely to any abnormality in infancy. The typical trigger finger in adults can practically always be associated with occupational or actual trauma, while in infants or children, before chronic trauma has altered the parts, some abnormality in the tendon mechanism must exist which would predispose the sheath and tendon to trauma. The main idea postulated as to setting up a trauma mechanism is the acuteness of the angle in the change of course or pull of the flexor pollicis longus at the metacarpopha-

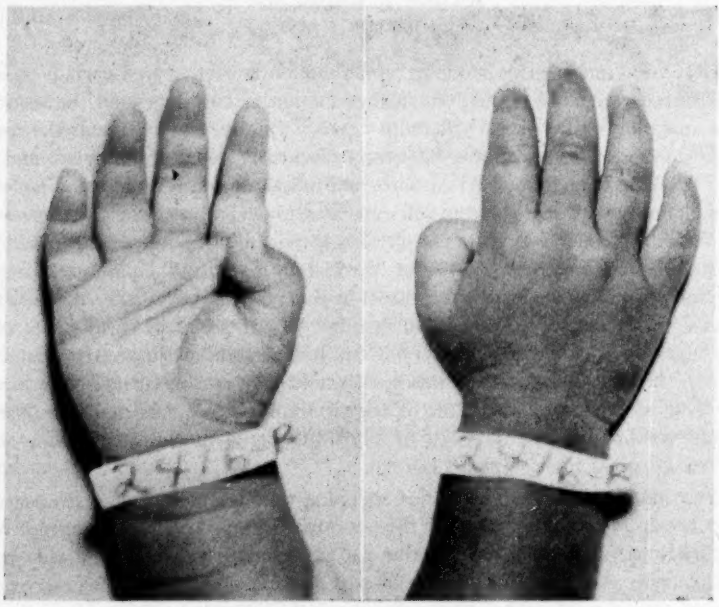

Fig. 1.—Thumb with distal phalanx locked in flexion.

langeal joint. If this were the only factor, the condition would be found much more frequently. It is felt, therefore, that there is a congenital abnormality that initiates the syndrome during the development of the fetus. The anatomic relationship of the flexor pollicis longus to the lateral head of the flexor pollicis brevis and to the adductor pollicis may be a factor. These muscles arise in somewhat parallel fashion. The flexor pollicis brevis inserts into the radial side of the base of the proximal phalanx of the thumb, and the adductor pollicis inserts into the ulnar side of the base of the proximal phalanx, with the long flexor passing between them. Possibly a band between these muscles, passing over the tendon sheath of the flexor pollicis longus, or an abnormally close association of the three structures may be the factor initiating the syndrome, although at the time of surgery nothing could be definitely seen substantiating this. A variation of the flexor pollicis brevis muscle is described

in Gray's Anatomy,[3] where it is shown that a portion of this muscle may be separate and insert into the ulnar side of the proximal phalanx of the thumb with the adductor pollicis. It is then described as the first volar interosseus muscle. To reach this insertion, the brevis muscle would cross under or over the flexor pollicis longus tendon.

In support of the congenital or hereditary background, in two of the cases reported in this paper there was a definite family history of the condition. The other five patients all came from the same island in the Hawaiian group: Kauai. All five of these patients also had a Filipino ancestry. It was considered strange that there should be so many cases from the one island. Possibly the condition was just being recognized as existing there, having the same incidence in other islands without being recognized.

In this paper we report seven cases. In two the condition was bilateral; in three it involved the right thumb, and in two it involved the left thumb. The patients' ages at the time of surgery ranged from 15 months to 6 years. Jahss's [4] statement

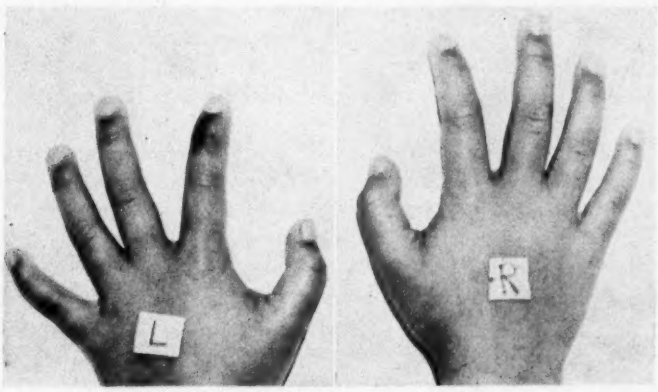

Fig. 2.—Bilateral trigger thumbs.

in his paper that the statement of the mother as to the time of the onset of the condition cannot be entirely relied on holds true in this series, and this is probably because the natural position of the infant's hand is somewhat clenched, with the thumb held in the palm. The deformity is frequently not noticed until the child begins to grasp objects. In our series the condition was recognized before the age of 1 year and most frequently at about 6 months of age. Sprecher,[5] in his series of 12 cases, reported that in four the condition was noticed immediately at birth. In two of our cases, the history was given that the thumb could at first be fully extended after a snap at about 150 degrees, but soon the thumb locked with the distal phalanx in flexion. In all the other cases the patients were unable to extend the thumb beyond 150 degrees from the onset or when the condition was first noticed.

3. Gray, H.: Anatomy of the Human Body, edited by W. H. Lewis, Ed. 23, Philadelphia, Lea & Febiger, 1936.

4. Jahss, S. H.: Trigger Finger in Children, J. A. M. A. **107**:1463, 1936.

5. Sprecher, E. E.: Trigger Thumb in Infants, J. Bone & Joint Surg. **31A**:672 (July) 1949.

The child does not show signs of pain unless forced extension is attempted. Examination shows a definite firm nodule over the tendon sheath of the flexor pollicis longus at the metacarpophalangeal joint. This feels almost bony-hard in consistency or, as one author described it, feels like a sesamoid bone.[6] The distal

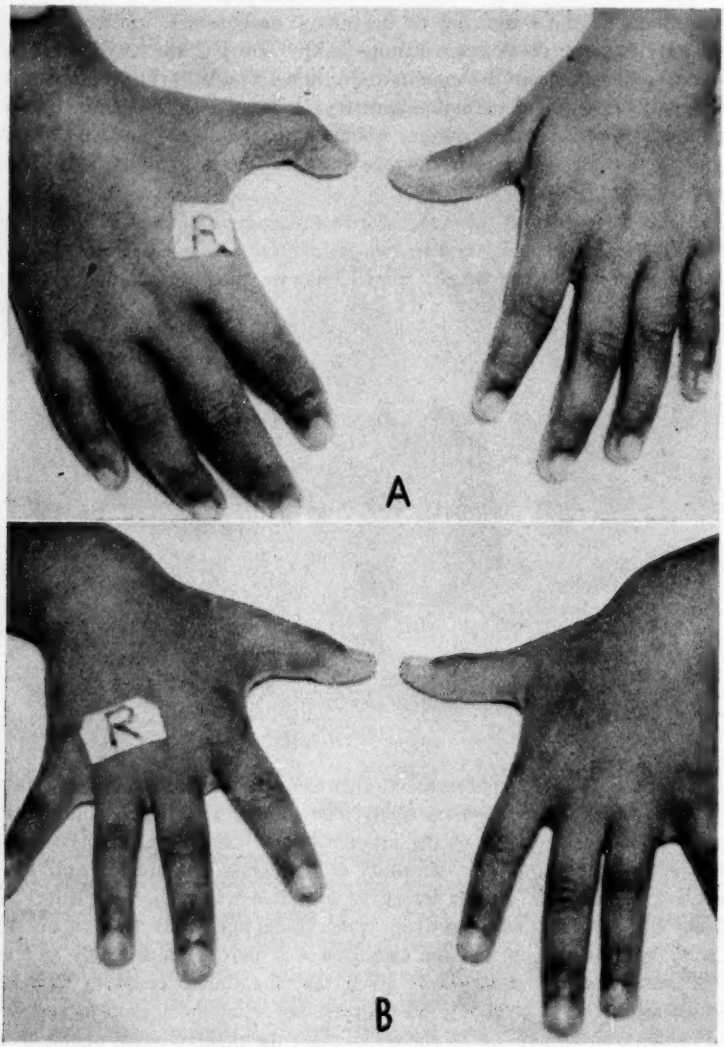

Fig. 3.—Trigger thumb of right hand; *A*, before operation; *B*, after operation.

phalanx is locked in flexion, because the stronger flexor, as compared to extensor pull, keeps the enlarged portion of the tendon on the proximal side of the constricting band.

6. Zadek, Isadore: Stenosing Tenovaginitis of the Thumb in Infants, J. Bone & Joint Surg. **24**:326 (April) 1942.

The pathology of this syndrome appears to be primarily in the tendon sheath and the tendon enlargement, a reaction to pressure of the tendon attempting to pass through the sheath. In one of our cases the tendon appeared edematous. Microscopic study of the tendon sheath in one of our cases showed only hyperplastic fibrous tissue. Jahss,[4] in his article, described the pathological study of the tendon sheath as showing hyalinized connective tissue with fibrous villi. We found no adhesions or free fluid within the tendon sheath in our cases. There was a definite enlargement of the tendon proximal to the thickened sheath in all our cases.

The treatment consists simply of incision of the thickened sheath, completely decompressing this enlarged tendon. Prolonged stretching will, as described by Jahss,[4] stretch the anterior ligament at the metacarpophalangeal joint, with the resulting hyperextension of the joint. A portion of the thickened sheath may be removed, although this is not felt to be necessary. During the operation, as soon as the thickened area is cut the tendon is released, full extension is obtained, and final cure is delayed only by the wound healing. The skin incision is made transversely in the flexor crease over the nodule at the metacarpophalangeal joint and needs to be only about ½ in. (1.7 cm.) long. The resulting scar can hardly be seen after a few months. The tendon sheath must be adequately opened longitudinally. This can be determined by passing a probe up and down the sheath. Only the skin is closed in these cases. The only immobilization we have used is an elastic bandage after surgery and then an aluminum splint for a couple of weeks. As regards conservative treatment before surgery, however, we have no quarrel with those who would prefer a period of splinting to hold the distal phalanx in hyperextension in those few cases in which the actual locking has not occurred. We found it too difficult to employ in these very young infants.

The results have all been excellent, with no disability or recurrences. None is to be expected because it is felt the etiological factor is removed when the constriction is released.

SUMMARY

Nine instances of trigger thumbs in seven cases of the condition in infants and children are reported. The presence of a probable hereditary background with a congenital deformity is observed in these cases, with a definite hereditary influence in two instances. The condition may be present from birth on but is probably not recognized until the child begins to use his hands to grasp objects. The diagnosis is relatively simple when the condition is recognized, and the treatment is surgical incision of the thickened tendon sheath of the flexor pollicis longus muscle. Results are gratifying.

MIGRAINE IN CHILDREN

A Report of Fifty Children

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FROM A study of 600 adults with migraine [1] it was noted that the onset of headache frequently occurred in childhood and also that psychological factors played a prominent role in the production of the migraine attack. As a result of these observations a group of 50 children with migraine who were seen at the Headache Clinic of the Montefiore Hospital for Chronic Disease, were studied in an effort to evaluate the syndrome. A preliminary study has been published.[2]

By "migraine" we mean that form of recurrent headache which is characteristically unilateral, occurs in bouts usually associated with gastrointestinal and/or ocular symptoms against a background of relative well-being, and is often preceded by visual or psychological disturbances and followed by sleep. There is usually a history of similar headaches in the parents or other members of the family.

The underlying causes of migraine are unknown. However, the mechanism of the symptoms is fairly well established and may be explained on a vascular basis.[3] Initial vasoconstriction of the cerebral arteries produces visual and possibly other preheadache phenomena prior to the onset of the headache. This prodromal period is then followed by dilation and distention of cranial arteries primarily in the distribution of the external carotid artery. It is this arterial dilation with increased amplitude of pulsation that is presumed to cause the headache.

METHOD OF STUDY

Each of our 50 children was studied in the following manner: A case history was taken by questioning both parents and child separately. Thorough physical examinations were performed, both neurological and medical, with laboratory studies included as indicated. The latter included visual field determinations, electroencephalograms, and blood determinations. Whenever feas-

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From the Headache Clinic of the Division of Neuropsychiatry, Montefiore Hospital for Chronic Diseases (Adjunct Attending Physician [Dr. Krupp]; Attending Physician [Dr. Friedman]). Assistant Professor of Clinical Neurology, Columbia University College of Physicians and Surgeons (Dr. Friedman).

1. Friedman, A. P.; Karron, I., and von Storch, T. J. C.: Migraine Problem, to be published.

2. Katz, J.: Friedman, A. P., and Gisolfi, A.: Psychological Factors of Migraine in Children, New York J. Med. **50**:2269 (Oct. 1) 1950.

3. Ray, B. S., and Wolff, H. G.: Experimental Studies on Headache: Pain-Sensitive Structures of the Head and Their Significance in Headache, Arch. Surg. **41**:813 (Oct.) 1940.

ible, a battery of psychological tests and four or five psychiatric interviews were included. The mothers of the children who were studied from a psychiatric point of view were also interviewed by the psychiatrist and the social worker.

PRECIPITATING CAUSES

In the present study it was observed that while precipitating factors cannot always be determined, stress situations of various kinds are usually the principal exciting causes of the attack, the trigger, so to speak. Some of the numerous causes that may precede headache are fear of failure, disappointment, rejection, unusual stimulation, and anxiety-producing situations. For example, children frequently complained of having headaches on the morning before an important school examination. Attending the movies or watching television or athletic events was at times the exciting "trigger" factor. However, this may possibly be because these factors stimulate unconscious phantasies and not because they induce eye strain. Perhaps the most common incident preceding headache was a family argument, with the child in conflict with its parents or siblings. Frequently both mother and child would develop migraine during an argument. The inability to express and control hostile feelings toward loved ones is certainly a factor in many of the headaches. Some children had headaches in less hostility-producing situations, such as going to a party or playing ball. On the other hand, it is noteworthy that headaches disappear or decrease during summer vacations, when hostility-producing situations are considerably minimized.

CHARACTERISTICS

Of 50 children with migraine, 35% complained of headaches before the fourth year. Their ages ranged between 3 and 15 years. There was no great difference in the incidence in the two sexes. A history of headache was noted in 84% of the parents. Of this group about 70% gave a history of classical migraine.

In some children the headache would start without any of the usual symptomatic picture of migraine, the typical syndrome developing later. However, in the majority, the syndrome in children was similar to that in adults, except that in children the headache was less severe and abdominal complaints were more prominent (but no less frequent). However, it should be emphasized that children are familiar with abdominal pain but may well find it difficult to describe the intensity of headache.

Headaches were preceded in 40% of the cases by gastric, psychogenic, or visual symptoms. It was noted that as the children grew older they tended to complain more of throbbing and unilateral pain. The majority of the headaches were frontal, temporal, and retro-orbital. Associated with them were gastrointestinal disturbances, photophobia, and a variety of autonomic disturbances, including sweating, chilly sensations, pallor, etc. It was our impression that children with migraine have a history weighted with attacks of biliousness, abdominal pain, and vomiting. In some the gastrointestinal symptoms occurred apart from the headache, while in others it occurred as part of the attack.

PSYCHOLOGICAL FINDINGS

The children were usually of superior intelligence, but there was no single dynamic pattern, and all types of personalities were observed. There was, however, a predisposition in the group toward certain personality traits. These consisted

of sensitivity, cleanliness, thoroughness, the need for approval, and the need to take seriously such responsibilities as school and dress. These children were in the upper part of their class and were considered by both teachers and parents as good students, as well as orderly and reliable individuals. Despite their generally good performance, these children commonly presented feelings of inadequacy, excessive guilt, and a strong superego (conscious or unconscious conscience). The inhibition of aggressive thoughts and actions occurred frequently, but not universally. In those children in whom hostility was excessively repressed, there was an excessive need for approval by parents and teachers. It is not surprising, therefore, that headaches among these children were often, though not always, related to unexpressed hostility toward these figures of authority whose approval is so urgently needed. These observations were confirmed by psychological tests.

The personalities of these children are not specific for migraine and may in fact be present in children with other disorders or in children who are symptom-free. Practically all of the children studied gave some evidence of psychogenic symptoms such as nail biting (frequent), feeding disturbances, thumb sucking, hair pulling, temper tantrums, enuresis, phobias, or frequent nightmares. However, only about one-fourth could be called psychoneurotic. A diagnosis of primary behavior disorder or psychosis could not be made in any of the cases.

The personalities of the mothers proved an interesting field for study. Many had migraine and their personality traits were often similar to those of their children.

TREATMENT

Results of Symptomatic Treatment.—In migraine the response to treatment is best evaluated in terms of symptomatic relief (that is, treatment of a headache attack per se) and prophylaxis (prevention of the attack).[4] The results of symptomatic treatment in our cases were fairly good. Treatment of migraine in children and adolescents, as well as in adults, is a difficult problem and must be adapted to the individual patient.[5] The parents and children were impressed with the importance of taking medication early. Many parents are reluctant to give medication to their children (or to themselves) and wait until the pain becomes severe. At this stage any type of medication is less effective. Acetylsalicylic acid was adequate in only 28% of our cases. On the other hand, a combination of 1 mg. of ergotamine tartrate and 100 mg. caffeine (cafergot®) gave adequate results in 60% of our cases and was helpful in 85%. Ergotamine and caffeine must be given early in the headache phase. At first one tablet is used, and if the headache is not relieved in a half hour it is again given every half hour for about four doses. When the effective dose has been determined (e. g., two or three tablets), this amount can be taken at once. In younger children, one tablet is usually sufficient. Occasionally, there will be side effects, such as nausea, chilliness, and dizziness, particularly with the use of the larger dose. It has been found that belladonna alkaloids will cut down such side effects, and occasionally the dose may be decreased. In some cases, the side effects can be lessened by administering suppositories containing ergotamine tartrate and caffeine. These are also helpful in attacks that have an abrupt onset with an early development of nausea and vomiting.

4. Friedman, A. P., and von Storch, T. J. C.: Recent Advances in Treatment of Migraine, J. A. M. A. **145**:1325 (April 28) 1951.

5. Friedman, A. P.: Modern Headache Therapy, St. Louis, C. V. Mosby Company, 1951.

Prophylaxis.—Parents can usually be made to understand that migraine is closely related to emotional upsets because of what has been observed in their children and in themselves and because many of them suffer similar headaches. It must be borne in mind that while limiting the patient's activities and rearranging schedules to provide regular sleep and regular meals may reduce the frequency of headaches, such restrictions may create new personality and adjustment problems for the parent and child.

The importance of the emotional factors is discussed with the parents. They are urged to understand some of the aggravating circumstances of the child's headaches and the importance of avoiding overt anxiety or giving excessive attention during the headache attack. Parents are encouraged to combine warmth with realism in their attitudes toward their children. With such an approach, therefore, prophylactic therapy can be carried on during the attack as well as during the intervals between attacks. The parents are advised to minimize their own headaches in the presence of the child. This appears to be one of the most significant aspects of prophylactic therapy. In fact, the familial incidence of migraine may in part be due to this factor of exposure, rather than to heredity.

The use of a psychiatric case worker to help the mother, and thus to help the dren without endangering either their security or their self-respect. Most of the opportunity for emotional release. They were able to talk freely about their feelings for their children and to discuss their own headaches as well as those of their children without endangering either their security or their self-respect. Most of the mothers had considered their situations unique, and when they discovered that many other mothers are faced with the same problem, the knowledge tended to make them feel less isolated and to relieve their guilt. Although the mothers were made to feel that responsibility for treating their children was shared by the clinic, they proved better able to assume responsibilities on their own in connection with family problems. Emotional release and acceptance by the case worker almost invariably resulted in increased willingness to change their attitudes.

More intensive psychotherapy was suggested for those children in whom the neurotic symptoms were outstanding or whose headaches were frequent and overwhelming. It is quite certain that when the child's need for self-assertiveness and love is not understood by parents who are rigid and strict in their training, the chances of the child developing psychoneurotic and psychosomatic symptoms are much greater. It is still too early to report the results of psychotherapy. A long-term study with follow-up reports will be necessary. It was noticed, however, that many of the children in our series improved almost immediately as soon as psychiatric investigation was started, or when the parents were educated as to the nature of the disease. Failure of therapy may be the consequence of failure to orient the parents to the child's problem.

SUMMARY

Fifty children with migraine under the age of 15 were studied. An analysis was made of the following factors: precipitating causes, symptomatic picture, psychological factors, and response to symptomatic treatment.

No significant differences in migraine attacks in children and in adults were noted. The condition, however, appeared to be milder in children with a greater preponderance of gastrointestinal symptoms associated with the headache or migraine equivalents.

Psychological aspects of this disease in children are important. Various stress situations would seem to be the principal exciting causes.

Ergotamine tartrate and caffeine (cafergot®) can be effectively used for symptomatic relief of the attack, particularly if given early. Effective preventive therapy depends upon an understanding of migraine as a total reaction of the child to his environment.

Advice and editorial assistance were given by Dr. Theodore J. C. von Storch, Chief of the Division of Neuropsychiatry. Mrs. Violet Segal of the Social Service Department and Mrs. Elaine Factor, psychologist, also assisted with this paper.

ABSTRACT OF DISCUSSION

Dr. Samuel Livingston, Baltimore: Dr. Krupp and Dr. Friedman's paper was so clearly presented that it leaves very little room for discussion. I should like to state, however, that I was surprised to note the great number of children with migraine that these doctors have had the opportuinity to study. I am sure that all pediatricians see many children with headaches in their private practices. We only rarely, however, see the typical migrainous type of headache.

I should like to ask Dr. Krupp and Dr. Friedman several questions. First, how many of their patients had abnormal electroencephalograms? Second, I should like to know the incidence of epilepsy in the families of their patients. Third, I should like to know how many of their patients, if any, also suffered with frank convulsive seizures in addition to the migraine headaches. Fourth, did frank convulsive seizures develop later in any of their patients?

We have been interested in the problem of headaches for some time. During the past year we have had the opportunity to study, in our epilepsy clinic, 62 children who complained of recurrent headaches. Each of these patients was studied extensively for the usual causes of headaches. Electroencephalograms were done on each patient. We found that 15 (or 25%) of them had abnormal electroencephalograms such as are seen in patients with frank epilepsy. These 15 patients were treated with anticonvulsant medication. The headaches disappeared in 10 of these patients following the administration of adequate doses of diphenylhydantoin (dilantin®) sodium. The other five patients were not favorably affected by anticonvulsant therapy.

In conclusion I should like to state that a headache may be the only clinical manifestation of an epileptic disorder.

Dr. Arnold P. Friedman, New York: I wish to thank Dr. Livingston for his very provocative and succinct remarks. I think one of the first things to realize is that we have a headache clinic and Dr. Livingston has an epileptic clinic. A child with a convulsion does not usually come to the headache clinic. There is more possibility that such a child will attend an epileptic clinic.

In our group we noted that the incidence of epilepsy was nil in our 50 children, and that one parent had epilepsy. The electroencephalogram changes were not significant. As to postseizure headache and migraine, the probable mechanism is vascular in both, but it seems that in migraine the external carotid artery is more involved, whereas in postseizure it is more of an intracranial involvement.

Regarding the frequency of migraine in children, most pediatricians in our locality agree with Dr. Livingston. However, we do see a number of children with headache. Perhaps it can be explained by the fact that we have a headache clinic and hence we are bound to see the people with this complaint. Interestingly enough, since the paper has been written we have had 25 more children with migraine.

TRANSVERSE MYELOPATHY IN CHILDHOOD

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CURRENT impressions and present-day pediatric texts generally regard transverse myelopathy as primarily an infectious process, that is to say, as so-called "transverse myelitis." There is considerable evidence, however, that a vascular etiology is actually involved in many, if not the majority of, cases, although in others the correlation with acute infections is too striking to be coincidental. The purpose of the present article is to present 25 cases of transverse myelopathy in childhood, and to consider their implications as to etiology, prognosis, and plan of management.

The concept of a vascular basis for transverse myelopathy is actually not a new one; it was emphasized by Bastian [1] as early as 1886. Bastian subsequently proposed three possible causes for vascular thrombosis involving the spinal cord, namely, syphilis, arteriosclerosis, and infections (of the central nervous system or elsewhere).[2] This type of causation and the absence of leucocytic infiltration microscopically were also pointed out by Singer [3] in 1902, and Continental neurologists of the generation of Marie and Oppenheim were apparently in general agreement on the subject. Early case reports were made by Preobraschenski [4] in 1908 and Spiller [5] in 1909 of patients having what has come to be called a "syndrome of the anterior spinal artery" involving the pyramidal and spinothalamic tracts, but sparing the posterior columns. Several discussions of the problem, with similar case reports, have been presented in the American literature by Ornsteen,[6] Schaller,[7] and Hogan,[8] among others, and most recently by Steegman [9] in 1952.

From the department of pediatrics, Harvard University Medical School, and the department of medicine, The Children's Medical Center.

1. Bastian, H. C.: Inflammation of Spinal Cord, in Quain, R., editor: A Dictionary of Medicine, New York, Appleton-Century-Crofts, Inc., 1862, p. 1474.

2. Bastian, H. C.: Thrombotic Softening of the Spinal Cord: A Case of So-Called "Acute Myelitis," Lancet **2**:1531, 1910.

3. Singer, H. D.: The Pathology of So-Called Acute Myelitis, Brain **25**:332, 1902.

4. Preobraschenski, P. A.: Ein Beitrag zur Lehre von der akuten syphilitischen Poliomyelitis, Neurol. Centralbl. **27**:1069, 1908.

5. Spiller, W. G.: Thrombosis of the Cervical Anterior Median Spinal Artery, J. Nerv. & Ment. Dis. **36**:601, 1909.

6. Ornsteen, A. M.: Thrombosis of the Anterior Spinal Artery, Am. J. M. Sc. **181**:654, 1931.

7. Schaller, W. F.; Roberts, A. M., and Stadtherr, E. F.: Acute Myelitis (Myelomalacia): Syndrome of Occlusion of Anterior Spinal Artery at the Fifth Cervical Cord Segment, J.A.M.A. **99**:1572, 1932.

8. Hogan, B. W.: Acute Myelitis: Syndrome of Occlusion of the Anterior Spinal Artery at the First Thoracic Cord Segment with Softening of the Cord, U. S. Nav. M. Bull. **40**:175, 1942.

9. Steegman, A. T.: Syndrome of the Anterior Spinal Artery, Neurology **2**:15, 1952.

TABLE 1.—*Cases of Postinfectious*

Case	Yr.	Sex	Age, Yr.	Preceding Infection	Time Interval Before Onset, Days	Nature of Onset: Earliest Symptom	Nature of Onset: Fever?	Nature of Onset: Stiff Neck?	Motor Impairment: Weakness: Legs	Weakness: Abdomen	Weakness: Trunk	Weakness: Arms	Respiratory Distress?	Interval Before: Increased Reflexes	Interval Before: Ankle Clonus	Interval Before: Positive Babinski
1	1929	M	5	Measles	14	Pain in legs	NR	+	+	−	−	−	−	None	None	None
2	1936	F	8	Measles	7	Urine retention	+	+	+	−	−	−	−	None	None	None
3	1936	F	9	Measles	7	Urine incont.	+	0	+	−	−	−	−	2 mo.	2 mo.	<7 days
4	1948	F	7	Measles	4	Weak legs + arms	+	+	+	±	±	+	−	4 days	4 days	?
5	1942	F	11	Varicella	0	Paresthesias in legs	+	+	+	+	+	+	±	5 days	?	?
6	1942	M	8	Varicella	16	Weak legs	+	NR	+	−	−	−	−	None	None	None
7	1934	F	5	Vaccination	15	Weak legs	0	+	+	±	−	−	−	None	None	None
8	1951	F	11	Vaccination	17	Pain in legs	+	0	+	±	−	+	−	3 days	3 days	3 days
9	1942	M	8	Scarlatina	7	Pain in legs	+	NR	+	+	±	+	−	None	None	<3 mo.
10	1950	F	5	Streptococcal; upper respiratory tract	11	Pain in legs	0	0	+	+	+	−	±	3 days	7 days	7 days
11	1930	F	10/12	Otitis media	21	Weak legs	+	NR	+	−	−	−	−	(Early)	(Early)	(Early)
12	1947	F	5	Otitis media	18	Weak arms	+	+	+	+	±	+	−	1 day	<2 mo.	<2 mo.
13	1934	F	10	Upper respiratory tract	3	Pain in legs	0	0	+	−	−	−	−	4 days	None	None
14	1950	M	15	Upper respiratory tract	1	Pain in back	+	+	+	±	−	−	−	?	?	?
15	1951	M	10	Upper respiratory tract	5	Weak arms	+	0	+	+	+	+	+	2 days	None	?

* Explanation of abbreviations: NR indicates not recorded as present or absent
? indicates finding present, but timing uncertain
Perm. indicates permanent
N indicates completely normal
G indicates good
F indicates fair
P indicates poor
} Degree of recovery as defined in text

There are also available reports of transverse myelopathy associated with a wide variety of infectious diseases, mainly viral, including measles,[10] rubella,[11] mumps,[12] herpes zoster,[13] and pneumonia,[14] as well as with the use of smallpox [15] and antirabies [16] vaccine.

10. Senseman, L. A.: Myelitis Complicating Measles, Arch. Neurol. & Psychiat. **53**:309, 1945.

11. Morris, M. H., and Robbins, A.: Acute Infectious Myelitis Following Rubella, J. Pediat. **23**:365, 1943.

12. Lightwood, R.: Myelitis from Mumps, Brit. M. J. **1**:484, 1946. Silverman, A. C.: Mumps Complicated by a Preceding Myelitis: Report of Fatal Case, New England J. Med. **241**:262, 1949.

13. Lhermitte, J., and Bussiere de Robert, J.: L'hyperpathie thoracique et les modifications du régime des réflexes superficiels et profonds consécutives à la myélite zostérienne, Rev. neurol. **74**:85, 1942.

14. Suchette-Kaye, A. I.: Acute Transverse Myelitis Complicating Pneumonia, Lancet **2**:417, 1948.

15. Dixonn, J. G.: Myelitis Due to Vaccination, J. Neurol., Neurosurg. & Psychiat. **7**:18, 1944.

16. Ansell, I.: A Fatal Case of Myelitis After Anti-Rabic Vaccine, Brit. M. J. **2**:338, 1948.

*Transverse Myelopathy in Childhood**

Sensory Impairment						Predominant Lateralization		Sphincter Impairment Day of Onset		Recovery: Degree	Recovery: Duration of Impairment Before Recovery			Comment
Pain	Temperature	Touch	Position + Vibration	Sensory Level	Zone of Hyperesthesia ?	Motor	Sensory	Urine	Feces		Motor	Sensory	Sphincter	
+	—	—	NR	L-1	0	..	..	?	?		Died within 1 week			Removed from hospital against advice
+	NR	±	NR	Legs	±	..	..	1st	?	N	1 mo.+	?	8 days	.....
+	+	+	—	L-2	0	..	..	1st	Early	F	8 yr.	8 mo.+	1 yr.	Contractures of right leg
—	NR	—	—	—	0	..	..	1st	Early	G	1 mo.	....	15 days	? burn of hand 4 mo. later
+	+	+	NR	T-2	0	..	..	2d	Early	P	Perm.	Perm.	2 yr.	Epidural hematoma not causing pressure at laminectomy; sensory level later fell to T-12
+	±	+	NR	T-4	+	..	..	1st	Early	N	10 days	10 days	4 days	.....
+	NR	—	—	L-3	0	..	..	1st	Early	N	1½ mo.	?	15 days	.....
—	NR	—	—	—	0	R	..	4th	4th	N	21 days	....	10 days	.....
—	—	—	—	—	0	..	..	Early	None	F	16 mo.	....	Early	Required orthopedic surgery
+	NR	—	—	T-3	±	L	L	None	None	N	3 mo.	1 mo.+	....	.....
+	NR	NR	NR	Legs	0	..	..	Early	None	F	Perm.	<7 yr.	3 yr.	Required orthopedic surgery
—	—	—	—	—	0	..	..	3d	3d	G	2½ mo.	....	2½ mo.	Still wets bed at night
+	NR	—	—	L-1	0	L	L	2d	2d	G	2 mo.+	?	1 mo.	.....
±	+	—	—	T-12	0	..	..	1st	1st	F	Partial perm.	Perm.	Partial perm.	Can walk but does so poorly; repeated burns
+	+	+	NR	C-4	0	R	L	1st	1st	G	6 mo.	1½ mo.	1½ mo.	In respirator for 7 days; given ACTH for 21 days

Other instances have been described immediately following plausible vascular incidents, such as dissection of an aneurysm of the aorta [17] and aortic compression.[18] Drugs have also been incriminated, especially arsenicals. While in many cases in this last-named group the disease was undoubtedly due in actuality to syphilitic endarteritis, possibly with exacerbation following therapy, in at least one instance it is known to have followed use of neoarsphenamine in the absence of demonstrable syphilis.[19]

It is partly, of course, a philosophical question whether postinfectious disease is, in fact, due to action of an infectious agent on the spinal cord itself, via an undefined hypersensitivity reaction, or by induced vascular lesions, possibly in turn also associated with sensitivity. Important questions of management, including prominently the use of corticotropin (ACTH) or cortisone, hinge on this point. The group of patients presently reported, being heterogeneous with respect to time, physicians responsible, thoroughness of neurological examination and investigative measures, plan of treatment, and length of follow-up, does not permit a definite conclusion on

17. Scott, R. W., and Sancetta, S. M.: Dissecting Aneurysm of Aorta with Hemorrhagic Infarction of the Spinal Cord and Complete Paraplegia, Am. Heart J. **38**:747, 1949.

18. Antoni, N., and Lindgren, E.: Steno's Experiment in Man as a Complication in Lumbar Aortography, Acta chir. scandinav. **98**:230, 1949.

19. Blankenhorn, D., and Wolff, H. G.: Myelitis Following Administration of Neoarsphenamine, J. Lab. & Clin. Med. **33**:1165, 1948.

TABLE 2.—*Cases of Transverse Myelopathy*

Case	Yr.	Sex	Age, Yr.	Significant History Prior to Onset	Nature of Onset: Earliest Symptom	Fever †	Stiff Neck †	Motor Impairment: Weakness: Legs	Abdomen	Trunk	Arms	Respiratory Distress †	Interval Before: Increased Reflexes	Ankle Clonus	Positive Babinski
16	1931	F	12	.....	Pain in back	0	0	+	+	−	−	−	<9 days	<9 days	<9 days
17	1932	F	10	.....	Abdominal pain	0	0	+	+	±	+	−	1 mo.	1 mo.	3 mo.
18	1937	F	½	.....	Limp all over	+	+	+	+	+	+	+	?	None	?
19	1943	F	5	.....	Weak legs	0	NR	+	+	−	−	−	<1 mo.	<1 mo.	<1 mo.
20	1945	F	12	Heavy lifting at onset	Pain in back	0	+	+	−	−	−	−	None	None	None
21	1947	M	5	Mumps 2 mo. before onset	Weak legs	+	+	+	±	−	−	−	4 days	4 days	4 days
22	1948	M	5	Fall from sled to ice at onset	Pain in back	0	+	+	+	±	+	−	<10 days	<10 days	<10 days
23	1948	M	4	Vomiting + abdominal pain for 4 days	Urine retention	+	±	+	+	−	−	−	1 mo.+	None	2 days
24	1949	F	13	Chronic suppurative; otitis media	Pain in legs	+	+	+	+	±	+	−	14 days	None	4 days
25	1952	M	12	.....	Abdominal pain	0	+	+	+	−	−	−	<17 days	17 days	17 days

* Abbreviations same as in Table 1.
† This case followed so far for only 2½ mo.; recovery not yet complete, but continuing.

the points mentioned. It is nevertheless of considerable interest as emphasizing a probable vascular origin in many cases, a frequently benign prognosis, and the need for caution in interpreting the "results" of therapy.

PRESENTATION OF CASES

Twenty-five cases of patients with transverse myelopathy admitted to the Children's Medical Center since 1929 are presented. Table 1 includes 15 patients with recent or concurrent acute infections at the time of onset and Table 2 includes 10 patients in whom such findings were absent or more remote, including 2 associated with trauma. Sex (15 girls and 10 boys) and age distributions were not remarkable, except that while the patients ranged from 6 months to 15 years of age, only 2 were under 4 years. Cases of myelopathy associated with evident encephalitis, mainly in connection with common childhood diseases, have been excluded from the present study.

While it cannot be argued that all of the cases associated with infections were of "infectious" origin or that those lacking such association were necessarily vascular, comparison of the two groups is both valid and of considerable interest if this limitation is borne in mind. The initial presenting complaint was rather variable, but not significantly different in the two groups.

Presenting Symptoms	Postinfectious Cases	Other Cases
Pain in extremities	5	1
Pain in trunk or abdomen	1	5
Weakness of extremities	6	3
Retention or incontinence of urine	2	1
Paresthesias in extremities	1	0

*in Childhood Without Preceding Infection**

Sensory Impairment						Predominant Lateralization		Sphincter Impairment Day of Onset		Recovery: Duration of Impairment Before Recovery				
Pain	Temperature	Touch	Position + Vibration	Sensory Level	Zone of Hyperesthesia	Motor	Sensory	Urine	Feces	Degree	Motor	Sensory	Sphincter	Comment
+	+	+	NR	T-8	0	..	R	4th	4th	N	18 days	18 days	18 days	Laminectomy showed widened cord and dilated vessels at level of T-7
+	+	+	—	T-5	±	L	R	3d	3d	P	Perm.	Perm.	3 mo.	No recovery of pain or temperature sense; repeated burns
+	+	+	NR	T-10	±	R	..	2d	None	P	Perm.	Perm.	Perm.	In respirator at onset
+	NR	+	NR	T-10	0	..	..	?	?	N	5 mo.	1 mo.+	<1 mo.	.....
+	—	—	—	T-7	0	L	..	?	?	N	14 days	?	<2 mo.	Partial permanent loss of pain sense persisted
+	NR	+	—	T-8	+	..	..	3d	1st	G	1 mo.	?	<1 mo.	.....
+	+	+	NR	C-6	0	..	..	1st	1st	P	Perm.	Perm.	Perm.	Sensory level fell to T-10; repeated burns
Probably none						..	..	1st	1st	G	2½ mo.	....	1 mo.	.....
+	NR	+	+	T-5	+	R	..	3d	2d	F	?	2 mo.	?	Laminectomy revealed dilated vessels at T-4-6
+	+	—	—	T-4	+	R	L	1st	1st	G†	†	†	11 days	.....

Weakness of the extremities developed in all of the patients during the first few days. In six patients there was ascending spread of paralysis, sometimes approaching in rate and severity the Landry type, while in three instances the arms were involved first, followed by the legs.

Distribution of Weakness	Postinfectious Cases	Other Cases
Legs but not arms	9	7
Legs followed by arms	3	3
Arms followed by legs	3	0
Trunk	6	4
Abdomen	9	9

The weakness was usually at first flaccid in nature, but in all patients whose motor involvement persisted over one month it gave way to increased muscle tone, increased reflexes, positive Babinski signs, or other evidence of pyramidal tract involvement. Sensory and sphincter disturbances were also present in the majority.

Extent of Impairment	Postinfectious Cases	Other Cases
Motor + pyramidal tract signs + sensory + sphincter	6	8
Motor + pyramidal + sphincter	4	1
Motor + pyramidal + sensory	1	0
Motor + sensory + sphincter, with recovery in under 1 mo	4	1

There were probably no sensory losses in 5 patients, Cases 4, 8, 9, 12, and 23. The ages of these patients were 7, 11, 8, 5, and 4 years, respectively, and in the three youngest of these the sensory examination was recorded as not wholly satisfactory due to poor cooperation.

During the time period covered by the present report, one additional patient was admitted with what was thought at first to be transverse myelopathy. She was a young woman of 21 years with sudden onset of paresthesias in the legs

followed by rapid progression of symptoms to include fever, stiff neck, weakness in the legs and abdomen, and impairment of sphincter control. There was complete loss of all sensation, including position and vibration, below the seventh thoracic level with a zone of hyperesthesia above this level. The cerebrospinal fluid contained 10 mononuclear cells per cu. mm. and a total protein of 140 mg. per 100 cc. A myelogram showed no significant abnormalities. Increased reflexes, bilateral ankle clonus, and positive Babinski signs developed within the first two weeks followed by gradual motor, sensory, and sphincter recovery to almost normal state over a one- to four-month period. This patient's original illness was thus quite consistent with that in the other 25 cases presented. Two and one-half months later, however, left retrobulbar neuritis developed with almost complete loss of vision, again followed by gradual recovery. It seems likely at present that this patient has a disease of the multiple sclerosis-neuromyelitis optica group, emphasizing that this possibility must be borne in mind in the differential diagnosis of transverse myelopathy.

A single patient of the series reported was treated with 5 mg. of corticotropin administered intravenously on admission followed by 100 mg. intramuscularly daily over a 21-day period with gradual withdrawal. He recovered almost normal function, but his case permits no conclusions, since considerable recovery was usual in both postinfectious and "idiopathic" cases.

Degree of Recovery	Postinfectious Cases	Other Cases
Completely normal	5	3
Good (slight limp, minor sensory loss, etc., but no significant functional impairment)	4	3
Fair (walking with marked limp or with aid of apparatus, permanent hypesthesia, some but not constant sphincter control, etc.)	4	2
Poor (completely or largely immobilized, permanent anesthesia, only partial sphincter control of "automatic" type)	1 (+ 1 death)	2

While prognosis in the postinfectious and idiopathic groups was not significantly different, there was in both some correlation between the degree of eventual recovery and the time interval before maximal return of function was achieved.

	Greatest Interval Before Maximal Recovery Occurred	
Degree of Recovery	Postinfectious Cases	Other Cases
Completely normal	3 mo.	5 mo.
"Good"	6 mo.	4 mo.
"Fair"	3 yr.	Uncertain

Other features of the cases presented, which are of especial interest in connection with the question of pathogenesis, will be discussed below.

COMMENT

An understanding of the anatomical arrangement of the normal blood supply of the spinal cord is essential for evaluation of the possible vascular pathogenesis of transverse myelopathy. Many of the available presentations are largely erroneous, but accurate accounts have also been presented, the classical publication being that of Kadyi [20] in 1889. More recent discussions are those of Tureen,[21] Suh and

20. Kadyi, H.: Über die Blutgefässe des menschlichen Rückenmarkes, Lemberg, Gubrynowicz u. Schmidt, 1889.

21. Tureen, L. L.: Circulation of the Spinal Cord and the Effect of Vascular Occlusion, A. Res. Nerv. & Ment. Dis., Proc. **18**:394, 1938.

Alexander,[22] and Herren and Alexander.[23] (Fig. 1, reproduced from the last-named, shows the arrangement of the arterial circulation.) The salient facts are well outlined in these articles and will not be considered in detail in the present report. The most significant feature, briefly, is an arterial supply from one anterior and two posterior longitudinal trunks, which are filled by afferent radicular arteries entering along the nerve roots and derived from the vertebral, ascending cervical, intercostal, lumbar, iliolumbar, and lateral sacral vessels. This afferent supply is segmental in the embryo, but not in the adult or even in the newborn infant.[22] While considerable anatomical variation exists, there are usually approximately eight anterior radicular arteries of significant size, each one unilateral, and the others are functionally

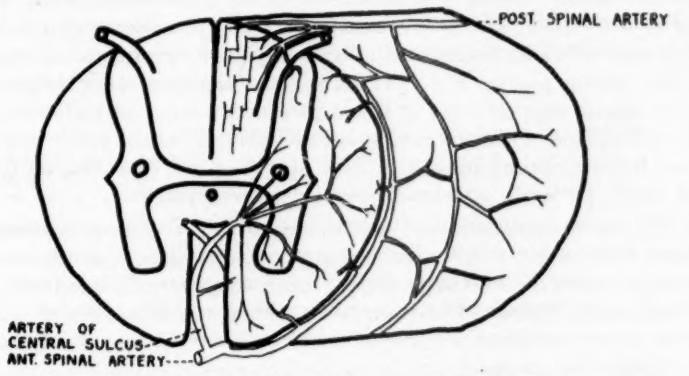

Fig. 1.—Afferent blood supply of the normal human spinal cord. (Reproduced from Herren, R. Y., and Alexander, L.: Arch. Neurol. & Psychiat. **41**:678, 1939).

unimportant owing to their small diameter. Suh and Alexander [22] report the number and diameter of the significant vessels in different regions of the cord as follows:

	Significant Anterior Radicular Arteries				Diameter of Anterior Spinal Artery, μ	
	Number		Diameter, μ			
Region	Adult	Newborn	Adult	Newborn	Adult	Newborn
Upper cervical	1	1	510	476	510	374
Lower cervical	1	1	578	306	442	442
Upper thoracic	2	1	374	272	374	342
Middle thoracic	1	0	340	...	340	272
Lower thoracic	1	2	510	476	578	374
Lumbar	1	2	872	748	1,122	600

Posterior radicular arteries are more numerous, perhaps 15 or 16 of significant size, but of smaller diameter than the anterior; the two posterior spinal arteries are also much smaller than the single anterior. Considerable circumferential as well as longitudinal anastomosis exists, but within the spinal cord most, if not all, arterioles are end vessels. The principal branch of the anterior spinal artery is the artery of the central sulcus, which passes up the central sulcus and then enters the gray matter of one side or of the other, but not usually of both, as it ordinarily

22. Suh, T. H., and Alexander, L.: Vascular System of the Human Spinal Cord, Arch. Neurol. & Psychiat. **41**:659, 1939.

23. Herren, R. Y., and Alexander, L.: Sulcal and Intrinsic Blood Vessels of Human Spinal Cord, Arch. Neurol. & Psychiat. **41**:678, 1939.

does not bifurcate. A number of penetrating branches are given off from the circumferential anastomotic vessels, but the artery of the central sulcus, together with branches directly from the anterior spinal artery, supplies most of the deeper cord, except for the posterior columns.

This situation is the basis of the so-called "syndrome of the anterior spinal artery," which is usually held to involve the pyramidal tracts and lateral and ventral spinothalamic tracts (although loss of pain and temperature may be dissociated from intact touch sensation) and to spare the posterior columns. Lesions in the artery of the central sulcus, or of slight longitudinal extent in the anterior spinal artery, may simulate the Brown-Séquard syndrome, except that there is no posterior column involvement. Under these circumstances one would expect motor weakness ipsilateral to the lesion, with contralateral loss of pain and temperature sensation, but sparing touch unless the lesion were of unusually great longitudinal extent, and in any event sparing position and vibration sense. Occlusion of the anterior spinal artery of several segments' extent would presumably affect several central sulcal arteries and produce a bilateral cord lesion with bilateral loss of motor function and pain and temperature sensation, with touch spared or not, depending on the longitudinal extent, but again with intact posterior column function.

In the case of traumatic cord lesions, the extent of sensory impairment has correlated quite closely with the accepted anatomical pathways,[24] and it would seem justifiable to expect the same in the present series of patients. "Dissociated" sensory loss (impairment of pain and/or temperature with or without touch, but with intact posterior column function) was frequent, and no less so in the postinfectious than in the "idiopathic" group.

Type of Sensory Impairment	Postinfectious Cases	Other Cases
Known to be dissociated (posterior columns spared)	6	4
Pain and/or temperature only	5	2
Pain and/or temperature + touch	1	2
Incomplete examination, but recorded data consistent with dissociated loss	5	4
Known not to be dissociated (also posterior column impairment)	0	1
No known sensory loss	4	1

The lateralization, complete or predominant, of the physical findings is also of interest because of the usual unilateral distribution of the artery of the central sulcus. One patient (Case 19) showed a Brown-Séquard syndrome, except that the status of position and vibration sense is not recorded. Others showed some degree of lateralization as follows:

Existence of Predominant Lateralization	Postinfectious Cases	Other Cases
Motor or sensory, but not both	1	4
Both, ipsilateral	2	0
Both, contralateral	1	2
No lateralization	11	4

Thus a total of 10 patients in both groups had dissociated sensory loss consistent with an "anterior spinal artery syndrome," including 6 after infections and 4 idiopathic cases. Three showed contralateral motor and sensory lateralization consistent with involvement of a single central sulcal artery. Only a single patient had

24. Boshes, B.; Brown, M., and Crouch, R. L.: Sensory Return in Partial and Recovery Spinal Cord Lesions, Neurology 2:81, 1952.

posterior column signs. Two cases of patients with ipsilateral motor and sensory impairment are impossible to explain fully at present. The remaining cases would seem to argue neither for nor against a vascular origin of the type under discussion, except that the posterior columns are known to have been involved in only one patient.

It is well known from existing reports that the direct and anastomotic blood supply of the human spinal cord is poorest in the upper and middle thoracic regions, and postmortem injection experiments indicate that injected material does not pass from below cephalad to this region.[22] An observation first made by Adamkiewicz,[25] and often reiterated subsequently, is that the majority of cases of human "transverse myelitis" have their levels in the upper and middle thoracic region of the cord. This is strikingly borne out among the "idiopathic" cases of the present series, but, in contrast to the other features previously discussed, the postinfectious cases are in this respect at sharp variance with the others.

Sensory Level	Postinfectious Cases	Other Cases
C-4 to C-6	1	1
T-2 to T-10	3	8
T-12 to L-3	7	0

The antecedent history of trauma just prior to onset of symptoms in Cases 18 and 22 is striking. That it does not necessarily imply a nonvascular origin is indicated by the reports of Spiller [5] and Grinker and Guy [26] of patients with similar histories of trauma who were shown at autopsy to have thromboses of the anterior spinal artery.

Dilated and tortuous posterior spinal vessels, not identified as venous or arterial, were described at laminectomy in Cases 16 and 24. Conceivably, these might have been secondary to a thrombosis elsewhere, or they may have represented vascular anomalies of the type described in detail by Wyburn-Mason [27] and Buchanan and Walker.[28] In this connection, the report of Black and Faber [29] of a 9-year-old boy with a blood-vessel tumor of the cord and dissociated impairment of pain and temperature sensation is striking.

X-rays of the part of the spine presumably involved were made of all patients; none showed any significant abnormalities. Only one of the 25 patients reported died, and unfortunately no autopsy was performed on this child, who had been removed from the hospital against advice shortly before. While the literature contains numerous reports of clinical "anterior spinal artery syndromes," there are few cases confirmed by autopsy. Spiller's [5] original patient had a thrombosis of

25. Adamkiewicz, A.: Die Blutgefässe des menschlichen Rückenmarkes: II. Die Gefässe der Rückenmarksoberfläche, Sitzungsb. d. k. Akad. d. Wissensch. Math.-naturw. Cl. **85**:469, 1882.

26. Grinker, R. R., and Guy, C. C.: Sprain of Cervical Spine Causing Thrombosis of Anterior Spinal Artery, J. A. M. A. **88**:1140, 1927.

27. Wyburn-Mason, R.: The Vascular Abnormalities and Tumors of the Spinal Cord and Its Membranes, St. Louis, C. V. Mosby Company, 1943.

28. Buchanan, D. N., and Walker, A. E.: Vascular Anomalies of the Spinal Cord in Children, Am. J. Dis. Child. **61**:928, 1941.

29. Black, W. C., and Faber, H. K.: Blood Vessel Tumor of the Spinal Cord in a Boy Aged 9 Years, with Special Reference to a New Diagnostic Syndrome, J. A. M. A. **104**:1889, 1935.

this vessel from the eighth cervical to the first thoracic level and softening of the cord from the fourth cervical to the third thoracic level. Other reports of similar postmortem findings are those of Grinker and Guy,[26] three syphilitic patients of Margulis,[30] Vogel and Meyer [31] (with an additional finding of ascending degeneration of the posterior columns), and the recent one from the Massachusetts General Hospital.[32] In contrast, there is at least one report [33] of a patient with similar symptoms, but with round-cell infiltration at autopsy without evidence of any thrombosis.

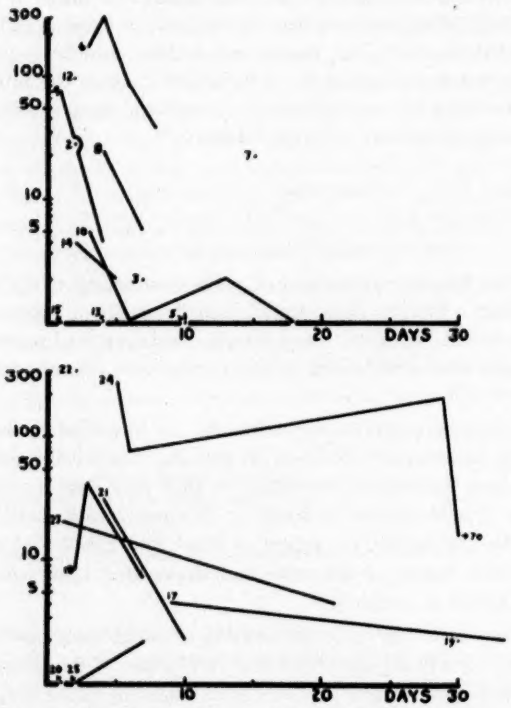

Fig. 2.—Cerebrospinal fluid cell counts (white blood cells per millimeter) of postinfectious and "idiopathic" cases of transverse myelopathy (by time after onset of symptoms), showing findings at initial and repeat lumbar punctures, if performed. Mononuclear cells predominated (70% or more) in all instances. Macroscopically bloody taps have been excluded. Top graph shows postinfectious cases; bottom graph shows other cases.

30. Margulis, M. S.: Pathologische Anatomie und Klinik der akuten thrombotischen Erweichungen bei spinaler Lues, Deutsche Ztschr. Nervenh. **113**:113, 1930.

31. Vogel, P., and Meyer, H. H.: Über eine akute Querlähmung des Rückenmarks und ihre anastomische Grundlage (Verschluss der vorderen Spinalarterie), Deutsche Ztschr. Nervenh. **143**:217, 1937.

32. Case records of the Massachusetts General Hospital, Case 38012, New England J. Med. **246**:30, 1952.

33. Hassin, G. B., and Broder, S. B.: Acute Myelitis, a Clinical-Pathological Study, J. Neuropath. & Exper. Neurol. **5**:106, 1946.

Spinal fluid cell counts and total protein determinations in the cases reported are presented graphically in Figures 2 and 3. Reports in the literature concerning the spinal fluid of patients in cases similar to the present group generally emphasize the finding of elevated protein without increase in cells.[34] The present series shows no significant difference in cellular findings between the postinfectious cases and the "idiopathic" group, although the counts were done at various stages of the disease, and the date after onset was sometimes not exactly recorded. Elevated

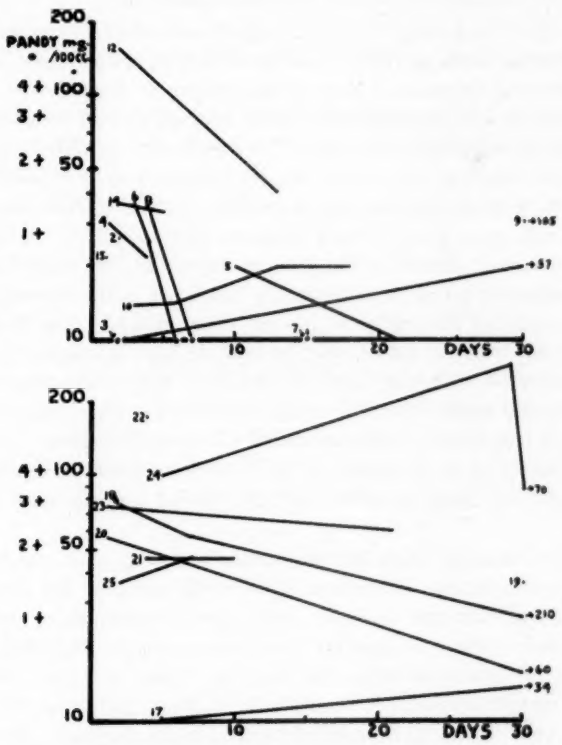

Fig. 3.—Cerebrospinal fluid total protein determinations of postinfectious and "idiopathic" cases of transverse myelopathy (by time after onset of symptoms), showing measurements in mg. per 100 cc., or degree of positiveness of Pandy test as a rough indication. Initial and repeat lumbar punctures, when performed, are included. Top graph shows postinfectious cases; bottom graph shows other cases.

proteins were commoner among the idiopathic group, as was a stiff neck or Kernig sign, while patients with preceding or concurrent infections more often had elevated

34. Jaffe, D., and Freeman, W.: Spinal Necrosis and Softening of Obscure Origin: Necrotic Myelitis Versus Myelomalacia; Review of Literature and Clinico-Pathologic Case Studies, Arch. Neurol. & Psychiat. **49**:683, 1943. Davison, C., and Brock, S.: Subacute Necrotic Myelopathy, J. Neuropath. & Exper. Neurol. **3**:271, 1944.

temperatures, as would be anticipated. (That the presence of fever and spinal fluid changes do not necessarily indicate infectious origin has been previously pointed out.[35])

Findings in Early Stage	Postinfectious Cases		Other Cases	
	With	Without	With	Without
Fever	11	3	4	6
Stiff neck or positive Kernig sign	7	5	7	2
Cerebrospinal fluid white blood count ≥ 10	6	7 (≤ 5)	6	2 (≤ 5)
Cerebrospinal fluid protein ≥ 35	5	7 (≤ 31)	7	1 (≤ 31)

(Reliable data were not available on some cases.)

Taken together, the findings indicate no significant differences between the cases of disease following acute infections and those lacking such a past history with respect to presenting symptoms, extent of impairment, or degree of recovery. The majority of both groups presented dissociated findings at least consistent with the so-called "anterior spinal artery syndrome," though limited by frequently incomplete neurological examinations, and several showed lateralization of similar implication. While spinal fluid cell counts showed in general no striking difference, the "idiopathic" group was more likely to have increased protein content. The location of the sensory level was usually in the lumbar region in the postinfectious cases, whereas the idiopathic group most commonly had levels in the thoracic level of the cord, corresponding to the region of poorest blood supply. The findings are in general consistent with the belief, current four decades ago and currently again becoming widespread, that many cases of so-called "acute transverse myelitis" are in reality of vascular origin frequently due to thrombosis of the anterior spinal artery. This is probably true of many cases associated with acute infections. The prognosis in children is likely to be favorable; 15 of 25 patients finally achieved degrees of recovery classified as "good" or better, and only 4 failed to obtain some useful return of function.

The probable vascular origin of many cases, including some classified as postinfectious or post-traumatic, casts some doubt on the rationale and probable benefit of corticotropin or cortisone therapy. It is quite conceivable, of course, and at present impossible to disprove, that the vascular lesion might take place on the basis of some form of hypersensitivity. The work of Kabat and his colleagues[36] on experimental encephalomyelitis in monkeys is of great interest in this connection. The studies involved material from killed tubercle bacilli, however; their interpretation would seem intimately bound up with complex problems of bacterial sensitivity, and their application to the problem of the human disorder now under discussion, highly uncertain. In any event, the predominantly favorable prognosis among children renders the evaluation of any type of specific therapy extremely difficult in view of the small numbers of such patients seen in even large medical centers.

35. Davison, C., and Keschner, M.: Myelitic and Myelopathic Lesions: A Clinico-Pathologic Study: I. Myelitis; II. Toxic Myelopathy; III. Arteriosclerotic and Arteritic Myelopathy, Arch. Neurol. & Psychiat. **29**:332, 600, and 702, 1933.

36. Kabat, E. A.; Wolf, A., and Bezer, A. E.: The Rapid Production of Acute Disseminated Encephalomyelitis in Rhesus Monkeys by Injection of Heterologous and Homologous Brain Tissue with Adjuvants, J. Exper. Med. **85**:117, 1947. Kabat, E. A.; Wolf, A., and Bezer, A. E.: Effect of Cortisone on Experimental Acute Disseminated Encephalomyelitis, Tr. Am. Neurol. A., p. 128, 1951.

SUMMARY

Twenty-five cases of transverse myelopathy in childhood are presented, 15 of which were associated with acute infectious diseases, while 10 lacked any such direct temporal connection.

The clinical resemblance of many of the cases of both groups to pathologically confirmed cases of thrombosis of the anterior spinal artery is emphasized. The findings in regard to dissociated sensory loss are similar in the two groups, but the location of the sensory level in the region of poorest blood supply (upper and middle thoracic levels of the cord) is more frequent among the patients without histories of concomitant infections.

The usually favorable prognosis in childhood, without specific therapy, is pointed out.

The implications of a study of these patients with respect to etiology, the use or nonuse of corticotropin (ACTH) or cortisone, and evaluation of any attempted therapy are discussed.

Permission to reproduce Figure 1 and data from Footnote 22 was obtained from Dr. Leo Alexander.

ORGANIC PHOSPHATE INSECTICIDE POISONING

Report of Two Cases Due to Parathion with Recovery in One

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ALTHOUGH preventive pediatrics is assuming increasing importance in the care of children, the incidence of accidental poisonings in childhood is by no means diminishing. Actually an absolute as well as relative increase in the incidence of such cases has been noted among patients admitted to the university service of the Grace-New Haven Community Hospital during the past eight years.[1] During 1951, 51 children were admitted to the pediatric service either because of actual poisoning or for observation after exposure to toxic substances; they constituted 5.1% of all admissions during the year.

Poisoning due to parathion, an organic phosphate insecticide, accounted for 2 of these 51 cases and was responsible for the only death due to poisoning during this time. This report is submitted because such cases present serious problems in diagnosis and therapy and have not heretofore been reported in the pediatric literature.

REPORT OF CASES

CASE 1.—M. M., a 25-month-old girl, was admitted to the university service of the Grace-New Haven Community Hospital because of sudden collapse with loss of consciousness.

Past History.—The child had been seen repeatedly in the outpatient department because of respiratory infections. She was admitted to the hospital for the first time at the age of 19 months because of severe growth retardation. Serum electrolyte and butanol-extractable iodine concentrations were normal, as were roentgenograms of the skull, wrists, and long bones. The fasting blood sugar was 85 mg. per 100 cc., and no reducing substance was demonstrated in the urine. She was discharged against advice after one day in the hospital with a diagnosis of probable primordial dwarfism.

Family and Social History.—The patient was the older of two children of a young gypsy couple who lived in three rooms together with three adult relatives. None of the adults were able to read or write English.

Present Illness.—The child had seemed well until an hour and a half before admission, when, while sucking a popsicle, she coughed, cried out, and then collapsed. The parents were

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Instructor in Pediatrics, Yale University School of Medicine (Dr. Chamberlin).

Assistant Professor of Pediatrics, Yale University School of Medicine, Markle Scholar in Medical Sciences (Dr. Cooke).

1. Lipton, B.: Accidental Ingestion of Poison by Children, Thesis, Yale University School of Medicine, 1951.

unable to revive the child and carried her to the emergency room. Despite careful questioning regarding possible exposure to drugs or poisons, including insecticides, no such information was obtained at this time.

Physical Examination.—The patient was a slim small girl weighing only 9.1 kg. who was limp and unresponsive. Her respirations were noisy and rapid, 52 per minute, but she was not cyanotic. The pulse rate was 150 per minute, the rectal temperature 99.14 F. (37.3 C.), and the blood pressure 140/40 mm. of mercury. The skin was moist, and the pupils were pinpoint in size. After partial dilation, using 1 drop of 1% atropine sulfate in each eye, normal fundi could be seen. There was much watery mucus in the mouth and over the tonsils and pharynx, but no evidence of inflammation. Loud coarse rhonchi were heard throughout both lung fields. The cardiac examination appeared unremarkable except for tachycardia. Intestinal peristalsis was hyperactive. The extremities were completely flaccid; no deep or superficial reflexes could be obtained.

Laboratory Findings.—The white blood count was 10,000 per cubic millimeter, with 40% polymorphonuclear cells and 60% lymphocytes. The hemoglobin was 13.5 gm. per 100 cc. Urinalysis revealed complete reduction of Benedict's solution, but no acetone was present. The blood sugar concentration was 170 mg. per 100 cc. (normal, 70 mg. per 100 cc.). The cerebrospinal fluid was normal. Cultures of the nose, throat, and blood were not remarkable. Roentgenograms revealed normal lung fields, cardiac silhouette, and abdomen, with no evidence of a foreign body in the respiratory tract. An electrocardiogram showed only tachycardia with the P-R interval at the upper limits of normal.

Course and Treatment.—Although a history of poisoning was lacking, the child's stomach was promptly lavaged with saline. Frequent suctioning of the nasopharynx was required. A slow venocylsis of interstitial salt solution [2] was started and 10 units of regular insulin were given subcutaneously.

At this time another relative appeared who stated that an insecticide had been sprayed in the patient's home about three hours before her collapse. He brought with him a large aerosol cylinder containing 10% parathion in 90% methyl chloride, manufactured by the Plant Products Corporation under the trade name of "plantthion." None of the relatives understood the significance of the warning labels.

The child was then given an injection of 0.20 mg. of atropine sulfate subcutaneously (twice the usual preanesthetic dose for her weight), and her body was carefully cleansed with soap and water to remove insecticide. Soon after the injection of atropine there was marked clearing of the respiratory secretions, so that suctioning was no longer necessary. The child seemed more alert and responded to painful stimuli, although no reflexes could be obtained. For the next six hours her status remained essentially the same, and it was thought that the critical period had probably passed. Two hours later, however, the child again became deeply comatose and repiratory secretions rapidly increased. As the special nurse prepared to suction, her breathing suddenly ceased. Artificial respiration and emergency stimulants failed to revive her, and she was pronounced dead 12 hours after admission. Permission for an autopsy could not be obtained.

CASE 2.—R. M., the 5-month-old brother of the above patient, was admitted to the hospital three days after the death of his sibling because of collapse and loss of consciousness.

Past and Family History.—The child had previously been well and the only significant family history was that included under Case 1.

Present Illness.—The patient was seen at the time of his sister's admission, although he allegedly had not been in the room which had been sprayed with parathion aerosol. Although his pupils were unremarkable and he appeared normal, he was given 0.06 mg. of atropine sulfate subcutaneously. The parents were given careful directions concerning decontamination of the room and exclusion of the infant from it. Nevertheless, 3½ days later he was brought to the hospital in a flaccid condition with the story of having suddenly become limp an hour previously. He had been well until this episode except for a period of prolonged crying two days before which had been attributed to constipation. Allegedly, at no time had he been in the room sprayed with parathion. His clean diapers, however, had been hanging in an open cloth bag

2. Electrolyte solution prepared by Sharp & Dohme Inc., for Dr. Daniel C. Darrow.

in this room at the time of spraying. Supposedly the entire room and all objects in it had subsequently been carefully scrubbed and aired. Investigation of the room suggested that these procedures had not been carried out as thoroughly as implied.

Physical Examination.—The patient was a limp, seriously ill, but well developed, 6.8 kg. infant. His respirations were noisy and rapid, 44 per minute, and his cry was weak. The pulse rate was 180 per minute, the rectal temperature 100.04 F. (37.8 C.), and the blood pressure 120/72. The skin was moist and the pupils were pinpoint in size, enlarging only slightly in near darkness. There was a watery rhinorrhea, and the mouth and oropharynx were filled with thin secretions, without injection of the mucous membranes. Loud moist rhonchi were present throughout both lung fields. The heart was unremarkable except for tachycardia, and the abdomen was normal. The extremities were completely flaccid; both deep and superficial reflexes were absent.

Laboratory Findings.—The white blood count on admission was 16,400 per cubic millimeter, with 71% polymorphonuclear cells, 27% lymphocytes, and 2% monocytes. The hemoglobin was 11.9 gm. per 100 cc., with 4,150,000 red blood cells per cubic millimeter. The urine was normal. Cultures of the nose, throat, and blood were unremarkable, as were fasting blood sugar and nonprotein nitrogen concentrations. The cerebrospinal fluid was within normal limits. The electrocardiogram on admission showed a biphasic T_3 and inverted T_2; subsequent electrocardiograms were normal.

A serum specimen obtained on admission was analyzed subsequently for the presence of parathion by a modification of the Averell-Norris method.[3] A value of three to four parts per million was obtained, but was not considered entirely reliable due to technical difficulties. No parathion was detected in a sample of whole blood obtained on the seventh hospital day.[4] Plasma and red blood cell samples obtained on the eighth day after exposure still showed a striking reduction of cholinesterase activity by the Michel method.[5] The plasma cholinesterase activity was approximately 20% of normal and the red blood cell activity 17% of normal.[6]

Course and Treatment.—On admission the child was given 0.24 mg. of atropine subcutaneously (three times the usual preanesthetic dose for his weight) and his skin was washed thoroughly. Oxygen was administered by face mask, and he was kept on the slide of an open tank respirator rather than in the usual hospital crib. Large amounts of watery mucus were obtained on frequent suctioning of the oropharynx. Members of the department of anesthesia assisted in determining the maximal safe atropine dosage.

Within 10 minutes of the initial injection of atropine the pulse rose to 210 per minute, there was a marked peripheral flush, and the copious respiratory secretions began to diminish. One hour later the mucous membranes were dry, the pulmonary rhonchi had disappeared, the pupils had dilated slightly, the deep tendon reflexes were present, and there were weak involuntary movements of the extremities. The pulse gradually fell to 130, but the activity of the deep tendon reflexes steadily increased until ankle clonus had appeared. Three hours after the first injection of atropine copious respiratory secretions suddenly reappeared, responding within 15 minutes to 0.1 mg. of the drug, which in this reduced dosage again produced transient tachycardia and flushing. The patient was now considerably more responsive; he sucked on a sugar nipple, and gazed about at those around him. The hyperreflexia with associated clonus persisted. His pupils were now normal in size and responded well to changes in light intensity, but reverted to nearly pinpoint size during sleep. Subseqently, atropine was administered as frequently as

3. Averell, P. R., and Norris, M. V.: Estimation of Small Amounts of O,O-Diethyl O,p-Nitrophenyl Thiophosphate, Analyt. Chem. **20**:753, 1948.

4. Dr. R. C. Hirt and Dr. P. R. Averell of the American Cyanamid Company performed these analyses.

5. Michel, H. O.: An Electrometric Method for the Determination of Red Blood Cell and Plasma Cholinesterase Activity, J. Lab. & Clin. Med. **34**:1564, 1949.

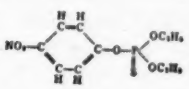
6. These determinations were kindly carried out by Dr. David Grob of the Johns Hopkins University School of Medicine through arrangements made by Dr. Joseph Guelich of the General Chemical Division, Allied Chemical and Dye Corporation.

needed to control excessive bronchial secretions, but the dosages required gradually decreased (Table). The infant received 0.79 mg. of atropine during the first 24 hours and a total of 1.64 mg. over a period of 4½ days.

Hyperreflexia with associated clonus disappeared after 24 hours. Lethargy gradually diminished during the next three days and parenteral administration of fluids was discontinued. Cold humidification was used to prevent damage to abnormally dry mucous membranes during the periods following administration of atropine, and penicillin procaine was given prophylactically throughout hospitalization. After one week the infant appeared normal, and he was discharged on the eighth hospital day. Follow-up physical and psychometric examinations have revealed no residuals.

Schedule of Atropine Sulfate Administration in Case 2

First 24 Hr.		Second 24 Hr.		Third 24 Hr.		Fourth 24 Hr.		Fifth 24 Hr.	
Hr. After First Dose	Dose, Mg.	Hr. After First Dose	Dose, Mg.	Hr. After First Dose	Dose, Mg.	Hr. After First Dose	Dose, Mg.	Hr. After First Dose	Dose, Mg.
0	0.24	25	0.05	51	0.025	73	0.05	106	0.05
3	0.1	27	0.05	53	0.025	89	0.05		
6	0.1	29	0.05	55	0.025				
8	0.1	31	0.05	58	0.05				
11	0.05	34	0.05	62	0.05				
14	0.05	38	0.05	65	0.05				
16	0.05	40	0.05	68	0.05				
19	0.05	42	0.05						
23	0.05	46	0.025						



Formula of parathion.

COMMENT

Parathion (p-nitrophenyl diethyl thionophosphate) (Figure) is one of several organic phosphates developed by German chemists as a result of research on war gases.[7] These agents were found to be the most effective insecticides yet developed.[8] Other chemicals of this group are di-isopropyl fluorophosphate (DFP), hexaethyl tetraphosphate (HETP), and tetraethyl pyrophosphate (TEPP).[9] Since its introduction into this country parathion, under many trade names,[10] has gained an important position in American agriculture. Its slow rate of hydrolysis and its

7. (*a*) Rohwer, S. A., and Haller, H. L.: Pharmacology and Toxicology of Certain Organic Phosphorus Insecticides: General Description of Their Activity and Usefulness, J. A. M. A. **144**:104, 1950. (*b*) Physicians and Phosphate Insecticides: Editorial, New York J. Med. **50**:1567, 1950. (*c*) Ward, J. C., and DeWitt, J. B.: Hazards Associated with Handling the New Organic Phosphates, Pests and Their Control **16**:34, 1948.

8. Grob, D.: Uses and Hazards of Organic Phosphate Anticholinesterase Compounds, Editorial, Ann. Int. Med. **32**:1229, 1950.

9. (*a*) Rohwer and Haller.[7a] (*b*) Grob, D.: The Anticholinesterase Activity in Vitro of the Insecticide Parathion (P-Nitrophenyl Diethyl Thionophosphate), Bull. Johns Hopkins Hosp. **87**:95, 1950.

10. Commercial Parathion: alkron,® niran,® thiophos.® Formulated Insecticides: "aphamite," "corothion," "edco 15," "genithion," "kilphos," "lethalaire G-54," "mackothion," "paradust," "paraphos," "penphos," "phos kit," "plantthion," and "vapophos." Parathion is known in Germany also as "E605," in France as "sulphos" or "SNP."

high solubility in lipids render it particularly valuable,[8] but it is these very properties which make parathion so dangerous. Thus severe poisoning may result from cutaneous absorption as well as from inhalation or ingestion. This absorption takes place without local inflammatory changes so that it remains entirely undetected until symptoms develop.[11] Moreover, the high lipid solubility of parathion probably leads to the binding of larger quantities in the lipids of the central nervous system.[9b] In addition it has been shown [12] that the oxygen analogue of parathion (in which an atom of oxygen is substituted for one of sulfur) has 300 times its anticholinesterase action in vitro and is about five times as toxic to experimental animals. There may be some oxidation of parathion to this more potent analogue in cases of poisoning with this agent.

The lethal dose of parathion for man is not known. Grob [13] has estimated from studies with DFP in experimental animals that the lethal dose of parathion for adults may be about 20 mg. intramuscularly, 100 mg. orally, and 500 mg. cutaneously.

Six other deaths due to parathion poisoning have been reported in this country. Three of these occurred in processing plants and three during spraying operations.[14] A seventh is reported to have occurred in Germany.[14b] There are no previous reports of exposures of children to parathion, but the death of a 10-year-old child almost immediately after ingestion of TEPP is briefly reported, and a second child is said to have developed severe symptoms after exposure to this compound.[14b] Three other deaths probably due to organic phosphate insecticides and more than 190 moderate to severe, but nonfatal, exposures in adults have been recorded.[14b] Many more fatalities may have occurred in Brazil where parathion is used extensively.[8]

The toxic action of these organic phosphate compounds is due to their anticholinesterase activity. The cholinesterases of the body comprise several types. Pseudocholinesterase (so-called because it can act on choline esters other than acetylcholine) hydrolyzes acetylcholine at the sites of muscarinic action (glands and smooth muscle) and is the type present in blood plasma. True cholinesterase hydrolyzes acetylcholine at the sites of nicotinic action (ganglia, skeletal muscle, and central nervous system) and is the type present in red blood cells.[15] These organic phosphates more effectively inhibit pseudocholinesterase,[15b] and therefore

11. Grob, D.; Garlick, W. L., and Harvey, A. M.: The Toxic Effects in Man of the Anticholinesterase Insecticide Parathion, Bull. Johns Hopkins Hosp. **87**:106, 1950.

12. DuBois, K. P.; Doull, J.; Salerno, P. R., and Coon, J. M.: Studies on the Toxicity and Mechanism of Action of p-Nitrophenyl Diethyl Thionophosphate (Parathion), J. Pharmacol. & Exper. Therap. **95**:79, 1949.

13. (*a*) Grob, Garlick, and Harvey.[11] (*b*) Grob, D.: Pharmacology and Toxicology of Certain Organic Phosphorus Insecticides: Toxicology; Council on Pharmacy and Chemistry, J. A. M. A. **144**:105, 1950.

14. (*a*) Grob, Garlick, and Harvey.[11] (*b*) Abrams, H. K.; Hamblin, D. O., and Marchand, J. F.: Pharmacology and Toxicology of Certain Phosphorus Insecticides: Clinical Experience; Council on Pharmacy and Chemistry, J. A. M. A. **144**:107, 1950. (*c*) Hamblin, D. O., and Marchand, J. F.: Parathion Poisoning, Am. Pract. & Digest Treat. **2**:1, 1951. (*d*) Grob, D.; Garlick, W. L.; Merrill, G. G., and Freimuth, H. C.: Death Due to Parathion, an Anticholinesterase Insecticide, Ann. Int. Med. **31**:899, 1949.

15. (*a*) Hamblin and Marchand.[14c] (*b*) Whittaker, V. P.: Specificity, Mode of Action, and Distribution of Cholinesterases, Physiol. Rev. **31**:312, 1951.

plasma cholinesterase activity falls before that of the red blood cells. Thus measurement of plasma cholinesterase activity becomes the most sensitive indicator of absorption of an organic phosphorus insecticide.[16] However, red blood cell cholinesterase activity more closely parallels tissue cholinesterase levels than does the plasma level, at least in dealing with recent exposures.[11] The development of toxic symptoms lags behind laboratory evidence of exposure. In fact, plasma cholinesterase may approach zero activity and red cell activity drop below 10% of normal before symptoms make their appearance. These symptoms, moreover, may disappear as the red cell activity increases above 15% of normal.[14c] Thus there is a considerable reduction of cholinesterase before symptoms develop and a small margin between levels producing no symptoms and those causing death.

The experiments of Freedman and co-workers [17] on rats exposed to DFP suggest that the severity of toxic symptoms closely parallels the level of brain cholinesterase. Symptoms first appear when the cholinesterase activity of brain has fallen to about 50% of normal. It has also been shown [18] in unanesthetized, curarized rabbits that an accumulation of acetylcholine in the brain is reflected in increasingly abnormal electroencephalographic tracings, but these abnormalities do not consistently appear until the cholinesterase activity has fallen to low levels. Furthermore Freedman and Himwich have demonstrated [19] a far greater susceptibility to DFP in the infant rat as compared to the adult, attributing this to the lower brain cholinesterase concentrations in the young of this species, thereby providing a smaller margin of safety. If this increase in susceptibility also applies to the human infant when exposed to parathion it may account for the severity of poisoning in our cases in the absence of signs or symptoms in the adults simultaneously exposed.

Since the adrenal medulla is stimulated by acetylcholine released by preganglionic fibers,[20] the tachycardia, hypertension, hyperglycemia, and glycosuria which were observed in Case 1 may be direct effects of the anticholinesterase activity of parathion. There are no other reports of hyperglycemia in organic phosphate poisoning, however, and both monkeys and dogs chronically exposed to DFP have shown no blood sugar abnormalities.[21]

It has been determined that red cell cholinesterase activity rises at a rate of about 1.2% per day (approximately the rate of erythrocyte synthesis).[14c] The red

16. Since the preparation of this manuscript a reasonably simple method for the detection in the urine of p-nitrophenol, an end product in the metabolism of parathion, has been developed. Waldman, R. K., and Krause, L. A.: A Rapid Routine Method for Determination of Paranitrophenol in Urine, Occup. Health **12**:37, 1952. This determination may prove to be of greater value in detecting exposure to parathion than the serum cholinesterase level.

17. Freedman, A. M.; Willis, A., and Himwich, H. E.: Correlation Between Signs of Toxicity and Cholinesterase Level of Brain and Blood During Recovery from DFP Poisoning, Am. J. Physiol. **157**:80, 1949.

18. Hampson, J. L.; Essig, C. F.; McCauley, A., and Himwich, H. E.: Effects of Di-Isopropyl Fluorophosphate (DFP) on Electroencephalogram and Cholinesterase Activity, Electroencephalog. & Clin. Neurophysiol. **2**:41, 1950.

19. Freedman, A. M., and Himwich, H. E.: Effect of Age on Lethality of Di-Isopropyl Fluorophosphate, Am. J. Physiol. **153**:121, 1948.

20. Best, C. H., and Taylor, N. B.: The Physiological Basis of Medical Practice: A Text in Applied Physiology, Ed. 5, Baltimore, Williams & Wilkins Company, 1950, p. 1085.

21. Koelle, G. B., and Gilman, A.: Relationship Between Cholinesterase Inhibition and the Pharmacological Action of Di-Isopropyl Fluorophosphate (DFP), J. Pharmacol. & Exper. Therap. **87**:421, 1946.

cell cholinesterase activity of 17% noted on the eighth day after exposure in Case 2 is consistent with this rate of regeneration. Plasma cholinesterase restoration is considerably faster and appears to parallel the rate of regeneration of new enzyme protein by the liver.[11] In a number of adult cases it averaged 14% on the first day, 9% on the second, and 2 to 6% thereafter.[8] Thus in our own case a plasma cholinesterase activity of only 20% eight days after exposure is lower than expected. Although our samples were frozen fairly promptly, it is possible that some activity was lost in transit. It is probable, however, that a 5-month-old infant is unable to regenerate plasma cholinesterase as rapidly as an adult. This slower rate of regeneration may be on a dietary basis, and since meat protein has been shown to accelerate plasma cholinesterase synthesis in rats,[22] meat should probably be fed during the recovery period even though the acceleration of tissue cholinesterase synthesis by meat protein has not yet been demonstrated.

One can only speculate on the mechanism of exposure of Case 2 to parathion, sprayed into a room which he allegedly never entered. All cooking utensils and other objects in the room were said to have been scrubbed and his milk came from freshly opened cans. The probable source of contact was the bag of diapers which, if sprayed vigorously, would allow percutaneous absorption of sufficient insecticide in the course of many diaper changes to produce severe poisoning. Since parathion is only 50% hydrolyzed after 120 days at 77 F. (25 C.),[8] an appreciable loss from these diapers would not have occurred in 3½ days.

Clinically, poisoning due to organic phosphate insecticides can be recognized readily and there is a characteristic progression of symptoms and signs. Since the muscarinic effects of acetylcholine predominate initially, nausea, diaphoresis, miosis, tenesmus, and excessive bronchial secretions are noted early, with progression to bronchoconstriction and pulmonary edema. Subsequently, as true cholinesterase becomes affected, nicotinic effects (muscular fasciculations and weakness) become prominent. As the acetylcholine concentration in the central nervous system rises central nervous system signs develop and ataxia, tremor, slurred speech, and coma with areflexia appear.[23] This combination of signs and symptoms is not seen in other clinical states. Of the cases of parathion poisoning thus far reported in detail all six patients who died and two who recovered went into deep coma with complete areflexia,[11] a picture apparently similar to that present in both our cases on admission.

Neostigmine (prostigmine®) bromide poisoning initially resembles intoxication due to organic phosphate insecticides, but it is of short duration because of the instability of the cholinesterase-neostigmine complex. In parathion poisoning, on the other hand, during the first hours after exposure there may be some restoration of cholinesterase activity due to a partial dissociation of the cholinesterase-parathion combination,[24] but this process subsequently becomes irreversible and the restoration of cholinesterase becomes dependent upon its resynthesis. Furthermore, in neostigmine poisoning involvement of the central nervous system is practically absent, presumably because of the relative insolubility of this compound in brain lipids in contrast to the organic phosphorus insecticides and particularly parathion.[9b]

22. Barrows, C., and Chow, B. F.: Federation Proc. 1952.

23. Grob, Garlick, and Harvey.[11] Grob.[13b] Hamblin and Marchand.[14c]

24. Footnotes 9*b* and 12.

The prolonged anticholinesterase effect of these organic phosphate compounds leads to a cumulative depression of tissue cholinesterase activity with intermittent exposures. This has been well demonstrated in regard to parathion, not only in animals,[25] but in humans as well. A large proportion of the fatal and severe cases [26] resulted from repeated daily exposures under working conditions. The degree of exposure on the final day was often no greater than that during the days or weeks preceding.

Unquestionably the most important factor in the successful treatment of organic phosphate insecticide poisoning is the use of frequent large doses of atropine for a prolonged period of time. Since dangerously low cholinesterase levels may persist for several days, premature cessation of atropine injections may result in a recurrence of severe symptoms. In fact, of the four previously reported fatal cases in which the patients had received atropine, three appear to have received only one injection [14c] and the fourth received no injections after the third hour of symptoms.[14d] In an earlier paper covering the organic phosphate insecticides the author stated that death in the majority of fatal poisonings occurred between 1½ and 4 hours after exposure,[27] whereas death actually occurred in the six cases reported thus far on an average of 9 hours after onset of symptoms [11] and more than 12 hours after onset in our own fatality. It is now believed that an adult showing signs of toxicity may need up to 2.0 mg. of atropine each hour, and possibly even more, to maintain constant complete atropinization.[28] Such large doses are usually well tolerated by these patients. Smaller doses should be continued for several days. Despite its great value in the treatment of these poisonings, atropine counteracts mainly the muscarinic actions of the anticholinesterase insecticides; it has no effect on their nicotinic actions [29] and relatively little [13b] on the central nervous system manifestations.

Besides the intensive use of atropine gastric lavage and cleansing of the skin should be carried out to remove as much unabsorbed insecticide as possible. Oxygen may be required, including the use of positive pressure should pulmonary edema develop. Artificial respiration and tracheotomy will be needed if the pharyngeal and respiratory muscles become paralyzed. Careful use of barbiturates may become necessary for the control of convulsions.[11] Morphine should not be administered. Likewise aminophylline and theophylline are contraindicated as bronchodilators in organic phosphate insecticide poisoning, since the ability of these drugs to produce cortical seizures was increased sixfold in the presence of DFP.[30]

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25. Footnote 12. Dubois, K. P.: Pharmacology and Toxicology of Certain Organic Phosphorus Insecticides: Pharmacology; Council on Pharmacy and Chemistry, J. A. M. A. **144**:105, 1950.

26. Footnotes 14*b* and *d*.

27. Lehman, A. J.: The Major Toxic Actions of Insecticides, Bull. New York Acad. Med. **25**:382, 1949.

28. Grob, Garlick, and Harvey.[11] Grob. 13[b]

29. Footnote 11. Goodman, L., and Gilman, A.: The Pharmacological Basis of Therapeutics: A Textbook of Pharmacology, Toxicology and Therapeutics for Physicians and Medical Students, New York, The Macmillan Company, 1941, p. 339.

30. Johns, R. J.; Bates, P. D., and Himwich, H. E.: The Effects of DFP on the Convulsant Dose of Theophylline, Theophylline-Ethylene-Diamine and 8-Chlorotheophylline, J. Pharmacol. & Exper. Therap. **101**:237, 1951.

Recent work in Switzerland [31] suggests that caramiphen hydrochloride (panparnit®) [32] may be a more effective antagonist to parathion than atropine, since it appears to counteract the nicotinic as well as the muscarinic effects of the anticholinesterase compounds. Extensive clinical trials of caramiphen in organic phosphate insecticide poisoning have not as yet been reported.

From the standpoint of preventive medicine it is recommended that closer supervision of the sale and distribution of these dangerous agents be carried out. The source of the aerosol cylinder which led to poisoning in our cases could not be determined. Thorough investigation by tracing the serial numbers revealed only that it had been sold to a large horticultural supply firm in New York City 10 months before. By restricting the sale of these chemicals the incidence of similar cases might be reduced.

SUMMARY

Two cases of poisoning in children are reported which were due to an organic phosphate insecticide (parathion). The pharmacology, diagnosis, and therapy of poisoning due to these agents are discussed.

31. Spindler, M.: Über Diäthyl-p-Nitrophenylthiophosphat, ein neues Schädlingsbekämpfungsmittel, Praxis **38**:936, 1949. Wilhelmi, G., and Domenjoz, R.: Therapeutische Möglichkeiten bei der Parathion-Vergiftung, Arch. internat. Pharmacodyn. **86**:321, 1951.

32. Available through Geigy Pharmaceuticals, Division of Geigy Company, Inc., New York.

Case Reports

ANTERIOR DISLOCATION OF FIRST CERVICAL VERTEBRA SIMULATING CEREBRAL BIRTH INJURY IN INFANCY

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RICHARD MASLAND, M.D.
AND
CARLTON HARRIS, M.D.
WINSTON-SALEM, N. C.

BECAUSE the pediatrician and the neurologist are so often confronted with the handicapped child showing evidence of paralysis and exhibiting seizures of one form or another due to cerebral birth injury, such signs and symptoms due to other causes are likely to be overlooked. It has long been recognized, but not sufficiently emphasized, that clinical pictures simulating cerebral injury can occur as the result of compression of the cervical cord. Compression of this type may be due to (1) tumors of the posterior fossa or the cervical cord or to bony exostoses in this location; (2) congenital anomalies of the region of the foramen magnum or the upper cervical spine, such as the Arnold-Chiari malformation or platybasia; (3) infections of the pharynx resulting in dislocations of the upper cervical spine,[1] or (4) traumatic dislocations and fractures of the upper cervical spine.[2]

It is apparent from case reports of children and of adults that the appearance of signs and symptoms resulting from chronic dislocation and fractures of the upper cervical spine may be delayed weeks, months, or occasionally years.[3] Such delay in occurrence of symptoms may be due to excess mobility of the spine which causes the spinal cord to undergo repeated trauma resulting in arachnoiditis, interference with the blood supply, or cystic degeneration; or it may be due to actual compression of the cord by bone or dura. It is essential that the possibility of such lesions being the cause of fainting attacks, paralyses, and apparent failure to develop

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From the departments of neurosurgery, neurology, and pathology of the Bowman Gray School of Medicine of Wake Forest College and the North Carolina Baptist Hospital.

1. Watson-Jones, R.: Spontaneous Hyperaemic Dislocation of the Atlas, Proc. Roy. Soc. Med. **25**:586-590, 1932.

2. Clark, W. A.: Fractures and Dislocations of the Cervical Portion of the Spine: With a Review of 89 Cases, Arch. Surgery **42**:537, 1941. Osgood, R. B., and Lund, C. C.: Fractures of the Odontoid Process, New England J. Med. **198**:61, 1928. Padula, R. D., and Tracey, E. J.: Fracture-Dislocation of Axis and Atlas in an Infant, J. A. M. A. **112**:1149, 1939.

3. (*a*) List, C. F.: Neurologic Syndromes Accompanying Developmental Anomalies of the Occipital Bone, Atlas and Axis, Arch. Neurol. & Psychiat. **45**:577, 1941. (*b*) Schwarz, G. A., and Wigton, R. S.: Fracture-Dislocations in the Region of the Atlas and Axis, with Consideration of Delayed Neurological Manifestations and Some Roentgenographic Features, Radiology **28**:601, 1937.

properly be kept constantly in mind. Otherwise the proper diagnosis will not become apparent until a surgically remediable lesion has brought about irreversible changes.

The following case report is given in some detail to further illustrate the importance of abnormalities of the cervical region as a cause of signs and symptoms resembling in many aspects those seen in cerebral palsy.

REPORT OF A CASE

R. B. K., a 21-month-old white boy baby, was admitted in extremis July 19, 1950, with a history of weak spells and difficulty in breathing for 10 months.

Past History and Present Illness.—The infant was delivered at full term, a spontaneous delivery Oct. 12, 1948, at a military hospital; the birth weight was 7 lb. 6 oz. (3,345 gm.). The mother had had no previous pregnancies and had no complications of this pregnancy. Anesthesia was caudal, after eight hours of labor. The mother suffered a postpartum hemorrhage, but was not seriously ill.

The infant appeared normal at birth; the head was not deformed, and there was no cyanosis, respiratory difficulty, or convulsions. He was a "feeding problem" until about 5½ months of age, but was strong, alert, and active during the neonatal period. At 3 months he began to hold up his head; he sat alone at 8 months, stood alone at 1 year, and took his first steps alone at 13 months. He began to speak single words at 10 months. The teeth appeared at the normal times, and there were no serious illnesses or injuries.

The first abnormalities were noted at the age of 10 months when, after the child had had an upper respiratory infection for about a week, he began to be fretful. Suddenly one night he was found to be extremely pale, listless, and cold. He was taken to a hospital, where he was immediately put in an oxygen tent and given fluids parenterally, as well as penicillin. The parents were told that the child had congestion of the lungs. He continued to cough for 10 days, but otherwise made a satisfactory recovery. Two weeks later another "seizure" developed, during which he became pale, listless, cold, and lifeless. This lasted about 10 minutes, and he was again taken to a hospital. On the basis of a single determination of the blood sugar, which was reported as 45 mg. per 100 cc., the parents were told the child was having attacks of hypoglycemia, and he was given a diet to correct this. During the next six months he had about three such attacks each month, most of them occurring in the morning. He remained conscious during such attacks, and after 10 minutes, usually after taking a bottle of milk, he was improved.

The mother had noticed at the age of 3 months that he did not use his left hand as much as his right, and when he began to walk, at 13 months, the right foot dragged and often struck against the left. He continued to use the left hand rather poorly, and his ability to walk, which improved from the age of 13 to the age of 15 months, began to deteriorate. Likewise, his weight, which had reached 21 lb. (9,525 gm.) at the age of 10 months, remained almost stationary to the age of 17 or 18 months. However, mental development continued normal, the child being able to name objects in magazines and appearing alert and intelligent.

At the age of 18 months he was seen by a pediatrician who told the parents the child had a "birth injury." The infant was taken to pediatricians and neurologists who concurred in this diagnosis and advised barbiturates. No roentgenograms of the skull or cervical spine were taken, although films of the chest and pelvis had been taken and reported as showing no abnormalities. Blood sugar at that time was reported as 90 mg. per 100 cc.

During the three months prior to admission to the hospital he showed progressive deterioration. He lost about 3 lb. (1,360 gm.) in weight, was lethargic and did not stand alone, had more frequent attacks of pallor, listlessness, and coldness of the skin, and began to exhibit occasional difficulty in breathing. He had five such attacks during the week before admission and was finally seen by a pediatrician who found him so gravely ill that he felt transportation of the child to another hospital would be dangerous. After the parenteral administration of fluids, the condition of the patient improved sufficiently to allow transfer to the North Carolina Baptist Hospital.

Physical Examinations.—Temperature was 99 F.; pulse rate, 100 per min.; respiration, 22 per min.; weight, 20½ lb. (9,300 gm.); height, 33½ in.; head, 19½ in.; chest, 17½ in.

The infant was acutely ill, breathing somewhat irregularly, but not cyanotic. The body was fairly well developed, but poorly nourished. Respiratory excursions were very slight and utilized the intercostal, diphragmatic, and accessory muscles of respiration. Flaring of the nares occurred with each inspiration. He was listless, held the arms and legs in a semiflexed position, and rarely moved spontaneously. Except for some slight inflammation of the pharynx, the general physical examination showed no abnormalities.

Fig. 1.—Lateral roentgenogram of cervical spine and base of skull showing anterior dislocation of the atlas on the axis and deformity of the nasopharyngeal air column.

Neurological Examination.—The head was normal in size. The anterior fontanel was closed. Cranial nerves, including the fundi, were entirely normal. The neck was not rigid, but there was some resistance to flexion. The chest was smaller than normal in circumference. He moved his extremities only rarely, and it was thought he moved the right arm more often than the left. He presented the appearance of extreme weakness and rarely talked or showed any spontaneous activity. He took fluids by mouth very poorly. However, it was observed that in spite of the child's critical respiratory weakness, he remained quite alert and attentive. He appeared to understand commands promptly, and, although motor performance could not be tested, it was the

impression of the examiner that his intellectual development was approximately normal for his age. The deep tendon reflexes, including those of biceps and triceps and knee and ankle jerks, were equal and active. They were not hyperactive. Abdominal and cremasteric responses were present and equal; Babinski responses were equivocal, but not clearly positive. Sensation over the entire body was apparently intact, although the patient's general condition did not permit a satisfactory evaluation of this aspect of the problem.

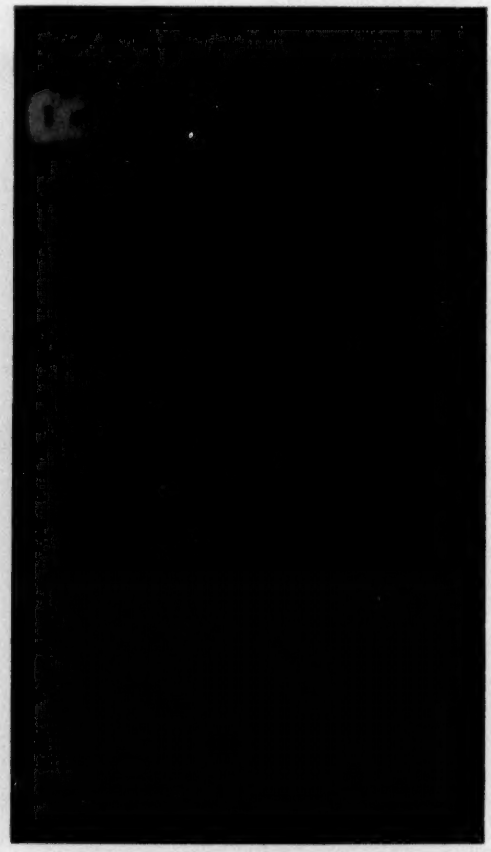

Fig. 2.—Posteroanterior roentgenogram of cervical spine with head rotated. Column of pantopaque (ethyl iodophenylundecylate) obstructed at first and second cervical levels. Patient in inverted position at time of exposure of film.

Laboratory Data.—Blood count and urine examination and tests for blood carbon dioxide and blood chlorides showed no significant abnormalities; tuberculin test was negative; Kahn test was negative; total protein of spinal fluid was 38 mg. per 100 cc.

Course in the Hospital.—Roentgenograms of the skull and chest showed no significant abnormalities. Films of the cervical spine demonstrated forward displacement of the atlas on the axis with an indistinct outline of the body of the atlas. There was deformity of the nasopharyngeal air column, thought to be due to a soft tissue mass (Fig. 1).

Puncture at the third lumbar level demonstrated a spinal fluid pressure below 100 mm. of water and a very slow rise and fall on jugular compression. The patient's condition did not

permit a thorough investigation of the cerebrospinal fluid dynamics. A dosage of 1.5 cc. of pantopaque (ethyl iodophenylundecylate) was introduced through the lumbar puncture needle, and the head of the fluoroscopic table depressed. The column of oil was followed to the foramen magnum and there at the first and second cervical levels demonstrated an apparent filling defect. A small amount of the opaque medium passed by this obstruction (Fig. 2).

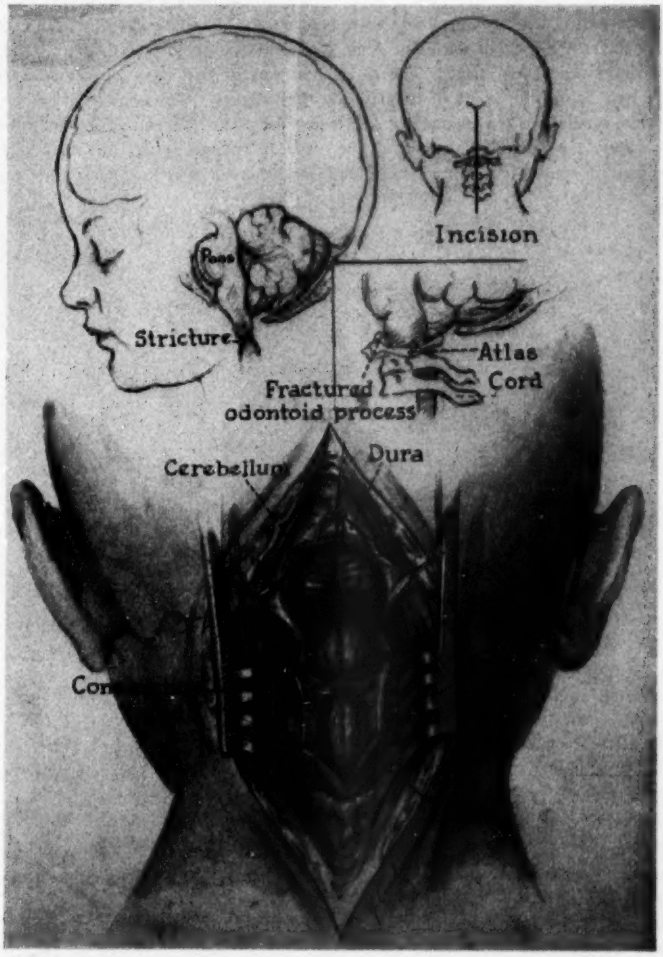

Fig. 3.—Artist's drawing of operative findings.

In view of the absence of any history of trauma and in consideration of the slowly progressive nature of the disability, it was felt that this child might have a dislocation of the atlas with encroachment on the cervical cord due to a tumor in that location. Before operation the left phrenic nerve was isolated and a fine wire wrapped around it. Stimulation of this with an electrophrenic stimulator gave a significant increase in respiratory excursion. Such stimulation was used repeatedly during the operation.

Operation.—The operation was performed on July 22, 1950. Ether anesthesia was administered endotracheally. A midline incision over the occipital bone and cervical spine was made with the patient in the prone position. The spinous process of the axis was prominent and the arch of the atlas displaced 1 to 2 cm. anterior to the position. The foramen magnum was enlarged by rongeuring a portion of the occipital bone, and the first and second cervical vertebrae were laminectomized. The dura was opened longitudinally in this exposure. There was a dense constriction of the dura with thickening of the dura beneath the arch of the atlas. When the dura had been opened, it was evident that there was a transverse indentation of the cervical cord immediately under the arch of the atlas (Fig. 3). Pressure on this was not relieved until the dural constriction had been opened. The cerebellum was normal, and the cervical spinal cord both above and below the point of constriction was slightly swollen. Introduction of a No. 27 hypodermic needle into the edematous area below the point of constriction was not successful in releasing any fluid from the substance of the cord. It was not cystic. The dura was left open and the wound closed in multiple layers with nonabsorbable surgical suture. The patient breathed

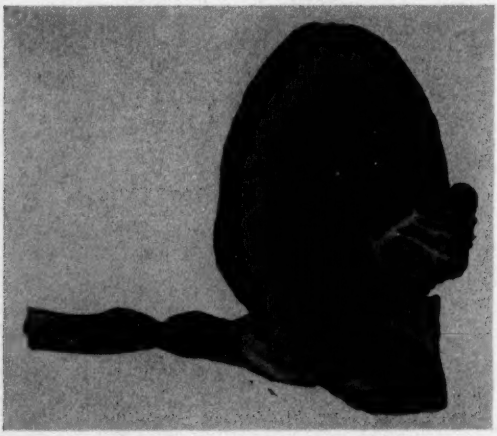

Fig. 4.—Sagittal section of brain stem, cerebellum, and upper cervical cord. Note constricted and degenerated area at first and second cervical levels. Weil myelin sheath stain (× 4.5).

irregularly after the operation, his condition being unimproved by the procedure, and in spite of electrophrenic stimulation and various respiratory stimulants, he died nine hours after operation.

Permission for a complete postmortem examination was obtained.

General Pathological Examination.—Except for body wasting and rather poor physical development, the examination of the body and its organs macroscopically and microscopically gave normal results.

Examination of the Cervical Spine and Central Nervous System.—Gross Findings: There was forward displacement of the atlas on the axis. The atlas and axis had been laminectomized and a portion of the occipital bone rongeured away. The odontoid process was in contact with the body of the atlas, completely separate from the body of the axis. The atlas showed no fusion with the skull, and no other bony abnormalities were found.

There was a constriction of the cervical cord at the first cervical level, reducing the circumference of the cord at this level to about one-fourth to one-fifth of its normal size. The brain was normal in its external appearance. After fixation in formalin for 10 days, coronal sections of the cerebrum were made at 0.7 to 1.0 cm. intervals. There was no dilatation of the ventricles, and no abnormalities were seen except for vascular congestion. The brain stem and the cere-

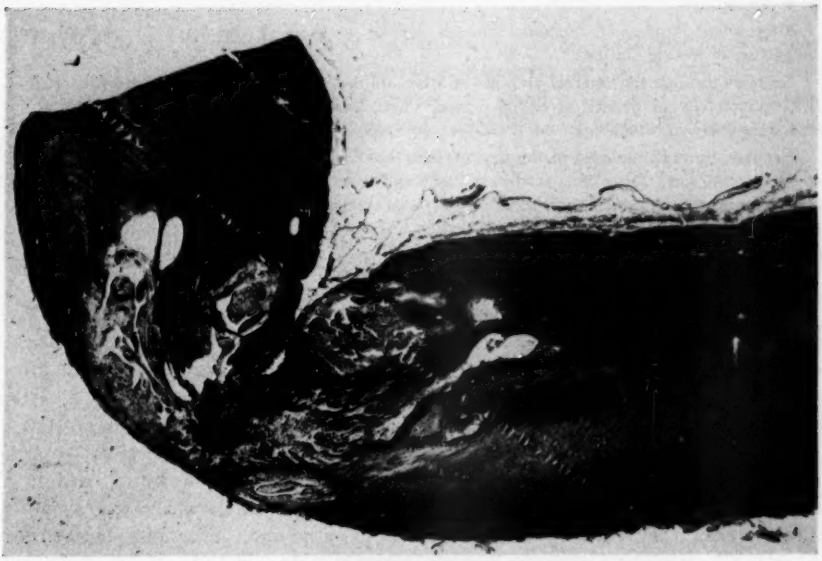

Fig. 5.—Sagittal section of upper cervical at point of constriction showing extensive scarring and cystic degeneration. Weil myelin sheath stain (× 4.5).

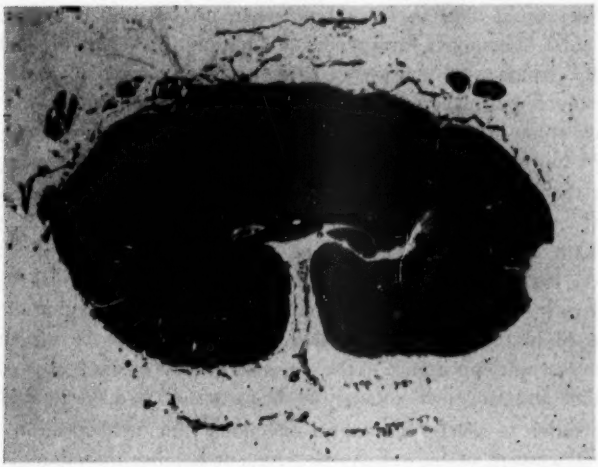

Fig. 6.—Cross section of spinal cord at fourth cervical level. There is relatively little evidence of descending degeneration. Weil myelin sheath stain (× 4.5).

bellum were sectioned in the sagittal plane together with the first four segments of the cervical spinal cord (Fig. 4). Coronal sections of one-half of the cerebellum and brain stem showed no abnormalities.

Microscopic Findings: Sections through various portions of the cerebrum showed some congestion of the capillaries.

Sections through the anterior arch of the atlas and the odontoid process disclosed the odontoid to be completely surrounded by fibrous tissue. There was no bone formation in the odontoid; it was completely cartilaginous, and there was no epiphysis at the base of the odontoid.

Sections through the cord at the first cervical level (Fig. 5) showed it to be about one-fifth of its normal size. There was moderate arachnoid reaction, and there were multiple cysts of small size in this region. There was marked degeneration of the cord in this location as contrasted with the slight descending degeneration distal to this point at the fourth cervical level (Fig. 6).

COMMENT

It is apparent from the roentgenographic findings, the operative findings, and the postmortem examination that the progressive downhill course of this patient during the last 10 months of his life was due to the chronic dislocation of the atlas on the axis with compression and degeneration of the cervical spinal cord. It is entirely likely that if the abnormality of the cervical region had been discovered a month before death, traction and reduction of the dislocation or surgical decompression might have been effective in restoring the function of the spinal cord to a relatively normal level. That the symptoms and signs in the early stages of the illness were those frequently seen in cerebral palsy no one could doubt, but as time went on the progression of the picture in the last three months of life suggested the need for a more thorough survey of the over-all problem.

An important clue to the noncerebral origin of this patient's disorder was to be found in the fact that he continued to show entirely normal mental development and a high degree of alertness at a time when progressive motor paralysis had reached the point of complete incapacity. In addition, hindsight reveals that during the "seizures," which were considered either epileptic or hypoglycemic, there had never been any impairment of consciousness. Roentgenograms of the skull and of the cervical spine in patients with problems as complex as were exhibited in this patient might more often disclose unsuspected and remediable pathology.

Whether the dislocation of the cervical spine resulted from trauma, infection, or congenital anomaly is not known. There is no history of trauma, but it is not known from the birth history whether the child was a vertex or breech presentation, and there is the possibility that trauma did occur to the cervical region during delivery. In view of the fact that symptoms of lesions in this region are not infrequently delayed in their appearance, this possibility cannot be discounted. The gross and microscopic appearance of the odontoid process do not serve to differentiate between trauma and congenital anomaly as the causes of the dislocation and fracture.

The association of severe pharyngeal infections with decalcification of the atlas and loss of attachment of the transverse ligament holding the odontoid process is well known.[4] Although this child did have an upper respiratory infection before the initial onset of symptoms, there is no indication that he suffered a retropharyn-

4. Titrud, L. A.; McKinlay, C. A.; Camp, W. E., and Hannah, N. B.: Non-Traumatic Atlanto-Axial Dislocation: Report of a Case with Recovery After Quadriplegia, J. Neurosurg. 6:174-180, 1949. Watson-Jones.[1]

geal abscess. Furthermore, the findings at postmortem examination showed the odontoid firmly attached to the anterior arch of the atlas. The findings favor trauma as the cause of the fracture-dislocation.

The evidence of degeneration of the descending tracts in the spinal cord below the first cervical level was slight but definite. However, as List [3a] and others have pointed out, the spinal cord may be reduced to a much smaller size than normal by chronic pressure without complete loss of function below the level of compression.

CONCLUSIONS

Chronic dislocation of the upper cervical spine with compression and degeneration of the cervical spinal cord may produce signs and symptoms which in the initial stages closely resemble those seen in cerebral birth palsy in infants.

A case is reported in which death resulted from anterior dislocation of the atlas with fracture of the odontoid process. The patient made a slow downhill course over a 10-month period, the diagnosis becoming apparent only at the terminal phase of the condition.

Roentgenograms of the skull and cervical spine in patients presenting a picture of seizures, paralysis, and physical retardation would be beneficial in discovering remediable lesions, such as in the patient reported herein, at a stage when therapy might be effective.

Progress in Pediatrics

AGENESIS OF THE LUNG

Report of a Case, with a Review of All Previously Reported Cases

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AND
P. H. HOLINGER, M.D.
CHICAGO

WE ARE reporting an additional case of an agenesis of the lung. A critical and complete review of all previously reported cases is presented to serve as a useful reference concerning this rare anomaly.

REPORT OF CASE

A 2-day old baby girl was seen by one of us (B. M. G.) because of a slight cyanosis and the possibility of a dextrocardia. The baby was born at Mount Sinai Hospital after an apparently normal course of labor, weighing 5 lb. 11½ oz. (2,595 gm.). The mother, tertigravida and tertipara, with two normal living children, was in good health throughout pregnancy and at the time of the birth of the infant. The infant was cyanotic at birth and did not breathe for several minutes until aspiration was done with a tracheal catheter and oxygen was supplied. Physical examination revealed what appeared to be a normally developed, though somewhat cyanotic, infant. Dullness and absent breath sounds were found on the right side of the chest, and the cardiac apex beat was felt in the right midclavicular line in the fourth interspace, but no heart murmurs were heard. The liver was palpated on the right side. An x-ray of the chest (read by Dr. J. Arendt, head of the x-ray department of Mount Sinai Hospital) (Fig. 1) revealed a homogeneous density throughout the right hemithorax. The heart shadow was displaced into that density and could not be distinguished. An x-ray of the spine and ribs (Fig. 2) revealed scoliosis of the lumbar spine and sacrum; the vertebrae appeared very irregular in shape, and the first right rib was malformed.

The blood examination revealed a red blood cell count of 5,050,000, hemoglobin 19.7 gm. per 100 cc., and white blood cell count 18,100. The differential count showed stab cells 1%, segmented cells 64%, eosinophiles 1%, lymphocytes 23%, and monocytes 11%. The color index was 1.25, and slight anisocytosis and a tendency to macrocytosis were reported.

An electrocardiogram (Fig. 3) ruled out a dextrocardia and showed a normal sinus rhythm, a rate of 185 per minute, a P-R interval of 0.08 second, a QRS of 0.05 second, a left axis shift, and no evidence of heart strain or chamber hypertrophy.

The striking physical findings, the homogeneous density of the entire right chest, the absence of heart murmurs, the associated abnormalities of the spine and rib, and the definite electrocardiographic findings of an absence of dextrocardia all pointed to the strong probability of an agenesis of the right lung, the clinical diagnosis made on the first examination.

Diarrhea developed on the second day after birth, and she was treated with streptomycin and procaine penicillin G by aqueous injection (crysticillin®). Because she was too weak to nurse she was fed by gavage. Respirations were rapid, varying between 60 and 70 per minute, and because of the cyanosis the baby was placed in an oxygen incubator. Although agenesis of the lung was considered to be the most probable diagnosis, several attempts were made to aspirate

From the Department of Pathology and Pediatric Cardiology of the Mount Sinai Hospital, and Chicago Medical School (Dr. Oyamada and Dr. Gasul); University of Illinois College of Medicine (Dr. Holinger).

the right bronchus by means of a tracheal catheter, but these attempts were unsuccessful. Two weeks after birth an attempt was made to inject iodized oil (lipiodol®) to visualize the bronchial tree, but roentgenograms could not be taken because the baby became extremely cyanotic. A bronchoscopic examination was made (by P. H. H.) at 18 days of age and the right main bronchus was found to be absent, confirming the clinical diagnosis. In addition, the left main bronchus was found to be stenotic. On the 25th day after birth, a temperature of 103 F. developed, respirations became noisy and labored, moist rales appeared over the left chest, and the patient died on the 28th day of life.

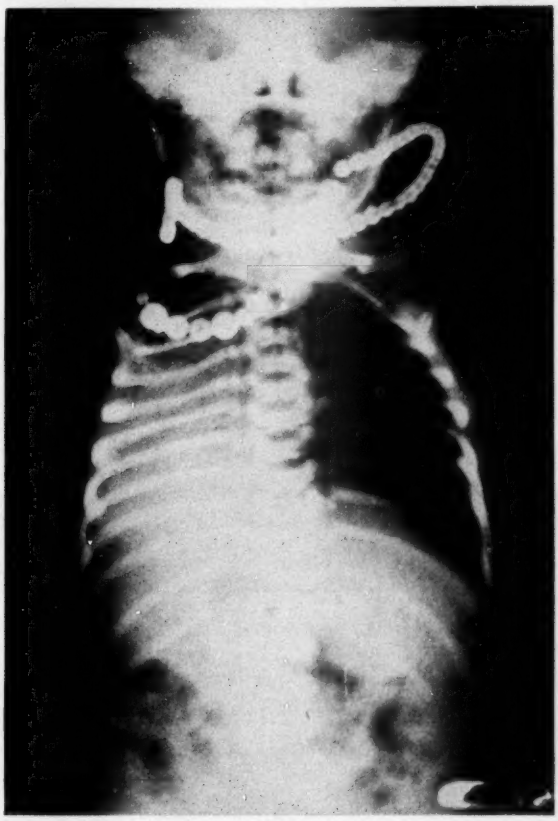


Fig. 1.—The right side of the chest is occupied by a homogeneous density. The heart shadow is on the right side and its borders cannot be made out accurately.

The postmortem examination showed a poorly developed but well-nourished baby girl weighing 2,933 gm. and measuring 30 cm. in the crown-rump and 47 cm. in the crown-heel lengths. The abdomen was moderately distended and contained 250 cc. of straw-colored fluid in the peritoneal cavity. The lower extremities presented no edema. The digits of both upper and lower extremities revealed moderate acrocyanosis. There were no external congenital anomalies.

The principle findings of interest were in the thorax. The chest wall was symmetrical. The diaphragm was more elevated on the right side, lying at the level of the fifth rib as compared to the level of the seventh rib on the left side. When the thorax was opened, the right chest cavity was obliterated by edematous velamentous adhesions resembling umbilical cord structure.

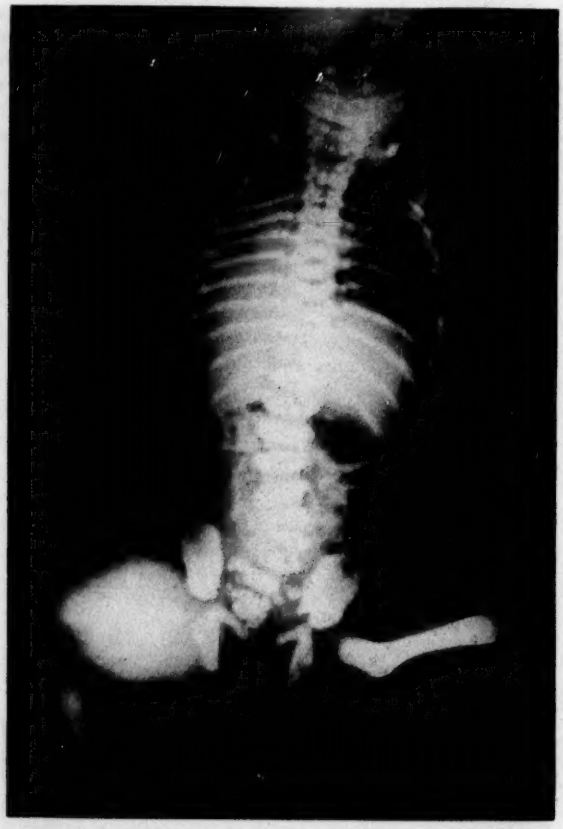


Fig. 2.—Malformed first right rib. Very irregular and malformed lumbar and sacral vertebrae.

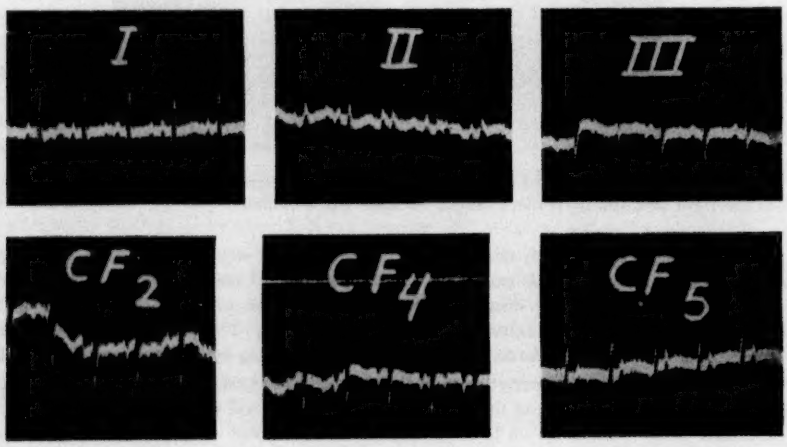


Fig. 3.—No evidence of dextrocardia; normal sinus rhythm; left axis deviation.

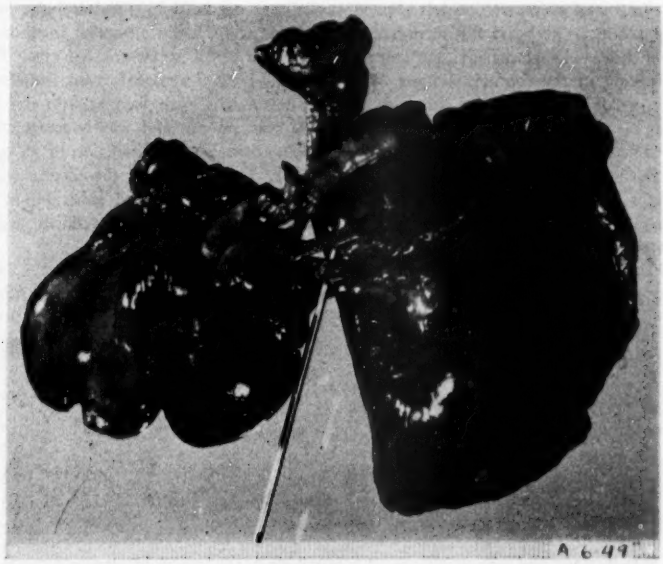


Fig. 4.—Thoracic viscera, anterior view. Only the two-lobed left lung was present, which received all the bronchial branches. The heart is shown to the left in this photograph, capped by the thymus. There were only a left pulmonary artery and left pulmonary veins; no trace of the corresponding right vessels was observed.

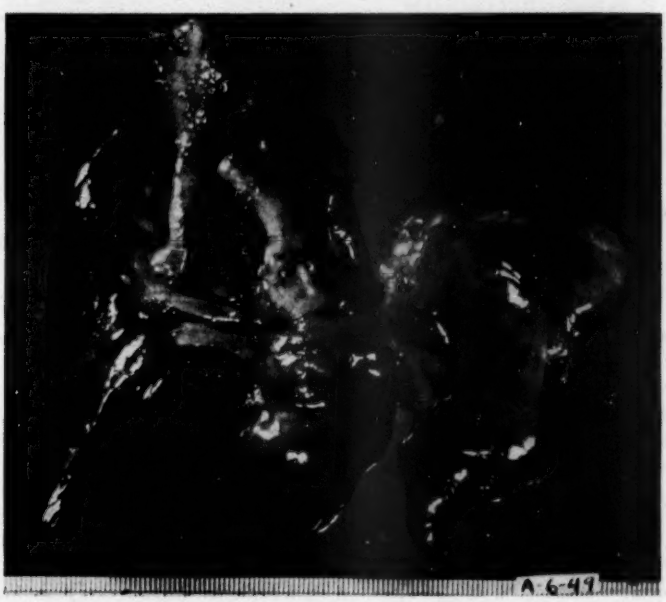


Fig. 5.—Thoracic viscera, posterior view. Here the entirely unilateral nature of the trachea and bronchus is emphasized. The soft tissue mass to the right of the heart is the thymus.

No right lung was present. The left pleural cavity was occupied by the left lung, which was free of adhesions. The heart and mediastinal structures were in their normal locations without any visible shift. The pericardial sac contained about 10 cc. of straw-colored clear fluid.

Examination of the thoracic organs (Figs. 4 and 5) after removal from the chest cavity revealed a small trachea 0.8 cm. in diameter and 8 cm. in length leading into the left lung. There was no right bronchus. The tracheal mucosa was smooth and was covered with frothy material. The left lung was composed of only one lobe, having a configuration of an upper lobe. It weighed 46 gm. (normal 54.7 gm. for both lungs). Over the apex the pleura was smooth and reddish. The consistency of the lung was firm over the apex and subcrepitant over the lower part, and the cut surface of the corresponding areas was moist and reddish, and pinkish-red, respectively. The right pulmonary artery was absent.

The heart weighed 30 gm. (normal 20.1 gm.) and had two small openings 0.5 cm. in diameter in the region of the foramen ovale. The ductus arteriosus was patent. From a normal-sized

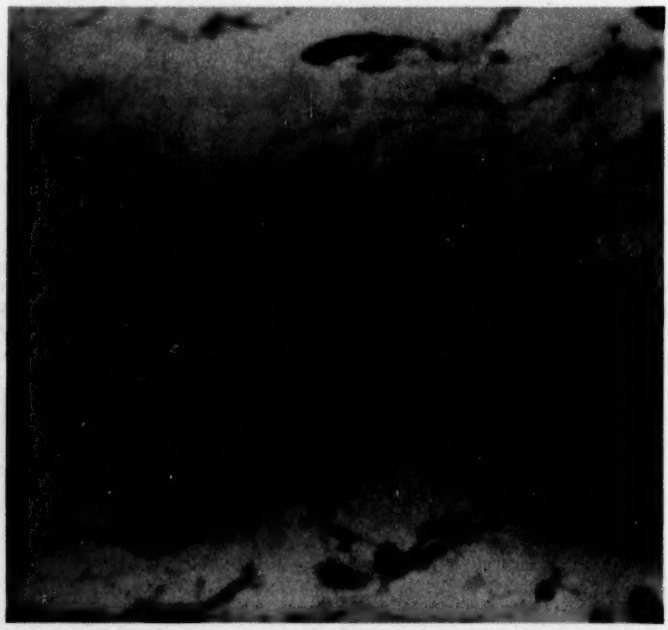


Fig. 6.—Photomicrograph of the embryonal connective tissue found in the right pleural cavity which contained no lung tissue. Note the myxomatous nature of this tissue (× 250).

pulmonary artery two smaller branches supplied the left lung. A single pulmonary vein returned from the left lung into the left auricle.

The thymus was normally placed but was partly embedded in the velamentous adhesions previously noted. There was no hypertrophy of the thymus. The kidneys and spleen showed acute passive congestion. There was a left-sided scoliosis of the lower spine and sacrum. The remaining organs demonstrated no gross changes.

The gross anatomical diagnosis was as follows: agenesis of the right lung with absence of the right bronchus; single-lobed left lung with atelectasis and bronchopneumonia; patent foramen ovale of the heart; patent ductus arteriosus; left-sided scoliosis of lower lumbar spine and sacrum; acute passive congestion of the kidneys and spleen; ascites.

Microscopic examination of the adhesions from the right pleural cavity showed a loose myxomatous-like embryonal connective tissue (Fig. 6) containing islands of fat cells and also more mature fibrous connective-tissue stroma. The left lung had areas of normally aerated

TABLE 1.—*Cases of Aplasia of One Lung Classes One and Two (Schneider)*

Author and Year	Age and Sex	Time of Diagnosis	Lung Findings	Associated Findings
Haberlein and Klinz 1787	20 yr. Male	At autopsy	Absent left lung, left bronchus, and left pulmonary vessels; left thorax filled with fluid; normal right lung	.....
Bell and Bell 1827	Young Male	At autopsy	Absent left lung, left bronchus, and left pulmonary artery; left hydrothorax, no inflammation; single pulmonary artery which was right vessel	Very small aorta
Bell and Bell 1827	24 yr. Male	At autopsy	Absent right lung, right bronchus, and right pulmonary vessels; trachea continued as left bronchus; single left pulmonary artery	.....
Heyfelder 1835	Newborn	At autopsy	Absent left lung; rudimentary left bronchus	Cyanosis
Hein 1837	6 wk. Male	At autopsy	Absent right lung and right pulmonary vessels; rudimentary right bronchus; pulmonary artery closed at base of heart; left lung supplied by ductus arteriosus	Interventricular septal defect; patent foramen ovale; cyanosis
Maschka 1862	2 hr. Male (Miscarried at 7 mo.)	At autopsy	Absent right lung, pea-sized saccular right bronchus, and absent right pulmonary vessels; unlobed left lung	Esophagus ended blindly at midpoint; atresia ani
Gruber 1870	Stillborn Female	At autopsy	Absent right lung, right bronchus, and right pulmonary vessels; unlobed left lung with only 1 pulmonary vein	.....
Herrero 1874	65 yr. Male	At autopsy	Absent left lung and left bronchus; undivided trachea continuous with right bronchus; right lung enlarged	.....
Stein 1878	6 wk.	At autopsy	Absent right lung and pulmonary vessels; rudimentary right bronchus	.....
Haberlein 1884	24 yr. Male	At autopsy	Absent right lung and right bronchus; left lung enlarged with 2 bronchi; received undivided pulmonary artery	.....
Théremin 1884	11 days Female	At autopsy	Absent left lung; small cartilaginous nodule at left bronchus site; trachea continuous with right bronchus; unilobar right lung	Pulmonary veins united to form large vessel that communicated with azygos vein; no pulmonary veins entered left auricle
Théremin 1884	127 days Female	At autopsy	Absent left lung, left bronchus, and left pulmonary vessels; right lung 2-lobed trachea continuous with right bronchus	Vein from right lung emptied into azygus vein
Gruber 1885	Fetus Female	At autopsy	Absent left lung, left bronchus, and left pulmonary vessels; right lung not lobed; received 3 bronchial branches	Patent ductus arteriosus; 1 pulmonary vein emptied into left auricle; large thymus; accessory liver and 3 accessory spleens
Tichomiroff 1895	24 yr. Female	At autopsy	Absent left lung, left bronchus, and left pulmonary vessels; right lung enlarged; received 3 bronchial branches	Large thymus; persistent left superior vena cava
Hanson 1901	15 min. Female Full term	At autopsy	Absent left lung, left bronchus 1/12 in. (0.2 cm.) long; right lung smaller than normal	Heart on right side; diaphragm lacking on left side; portion of small and large intestines in left pleural cavity
Findlayson 1902	Few hr.	At autopsy	Absent right lung and right bronchus; trachea ended in left bronchus; left lung hypertrophied	Undeveloped right pulmonary vessels
von Eiken 1907	Adult Female	Diagnosed malformation of pulmonary organs during life; more specific diagnosis at autopsy	Absent left lung; trachea shifted toward left; left bronchus ended blindly within left wall of trachea	.....
Oberwarth 1904	1 yr. Female	At autopsy	Absent right lung; right main bronchus ended blindly 0.5 cm. beyond bifurcation	Pulmonary vessels and associated congenital anomalies not described
Gross 1906	5 mo. 23 days Male	At autopsy	Absent left lung and left pulmonary vessels; blind left bronchus; right lung unlobed and enlarged	Left-sided hypoplasia of face, dermoid of left eye, anomalous right renal artery, and atresia ani
Ellis 1917	8 yr. Male	At autopsy	Absent left lung and left pulmonary vessels; right lung enlarged with 5 lobes	Hydropericardium; vegetations on mitral valve

TABLE 1.—*Cases of Aplasia of One Lung Classes One and Two (Schneider)—Continued*

Author and Year	Age and Sex	Time of Diagnosis	Lung Findings	Associated Findings
Tebbutt 1918	12 yr. Male	At autopsy	Absent left lung and left pulmonary vessels; main pulmonary artery divided into 2 branches before entering right lung	Heart in left thorax; endocarditis, mitral and aortic pericardial adhesions
Jarisch 1919	29 yr. Male	At autopsy	Absent left lung and left pleura; short left bronchial stump; right enlarged, imperfectly fissured; bronchopneumonia	Mediastinum shifted to left; heart against left chest wall
Levy 1920	49 yr. Male	At autopsy	Absent left lung, left bronchus, and left pulmonary vessels; right lung enlarged; pneumonia	Advanced syphilis of aorta and spinal cord
Muhamed 1923	19 yr. Female	At autopsy	Absent left lung and left bronchus; undivided trachea entered right lung, enlarged	Heart displaced to left; 2 thymus glands, 1 in left pleural cavity; post-traumatic rupture of liver and spleen
Dannheisser 1926	34 yr. Male	At autopsy	Absent left lung and left bronchus; right lung enlarged and received 2 bronchial branches; no fissuring of right lung	Heart displaced to left; large Meckel's diverticulum; anomalous kidney vessels
Heerup 1927	72 yr. Female	At autopsy	Absent left lung; right lung hypertrophic, extending toward other side	Mediastinal structures and heart shifted to left pleural cavity
Bönniger 1928	37 yr. Male	At autopsy	Absent right lung; wide right bronchus for 12 cm. beyond bifurcation, ending blindly as 2 small sacs; smaller bronchial branch curved behind, not clearly seen on x-ray	Scoliosis of upper thoracic spine; pulmonary vessels not described
Gilkey 1928	6 mo. Male	During life	Absent right lung	Mediastinum and trachea displaced to right; right radius congenitally absent
Welsch 1928	58 yr. Male	During life	Absent right lung; right pulmonary vessels and right vagus nerve; hypertrophied left lung with pneumonia; 2 tracheal branches to left upper lobe; 1 branch to left lower lobe	Heart and mediastinum displaced to right; increased number of tracheal cartilage rings; slight thoracic scoliosis
Bönniger 1931	27 yr. Male	At autopsy	Absent right lung; right bronchus ended as sac-like structure 4 cm. from bifurcation; trachea and left bronchus widened	Atrophy of right side of thorax; pulmonary vessels not described
Goehle 1928 Mitchell 1930 and 1946	2 yr.	At autopsy	Absent right lung, right bronchus, and right pulmonary vessels; cartilaginous stump at right side of site of bifurcation; left bronchus blocked by foreign body (chewing gum and peanuts); left lung hypertrophied	Heart in lower ⅔ of right pleural cavity; large thymus
Lochow 1928	5 mo.	At autopsy	Absent left lung and left bronchus; right bronchus continuous with trachea; absent left pulmonary vessels	Associated anomalies not mentioned
Finner 1932	22 mo.	At autopsy	Absent right lung, right pulmonary artery, and right bronchus; left lung emphysematous; peanut in trachea cause of sudden death	Heart and mediastinum displaced to the right
Minne and Gernez 1933	Stillborn Female	At autopsy	Absent left lung, left bronchus, and pulmonary vessels; right lung enlarged with no fissures; trachea continued directly into right main bronchus having 2 branches	Heart filled left chest; anencephaly; atrophic adrenals
Hepner 1934	3 mo. Female	At autopsy	Absent left lung and left pulmonary vessels; right lung incompletely fissured and enlarged	Heart filled left thorax; left diaphragm high; accessory spleen
Knott 1934	5 wk. Male	At autopsy	Absent right lung and right bronchus; left lung hypertrophied; bronchopneumonia	Dextrocardia; mediastinum displaced to right
Dyson 1934	6 mo. Female Premature	At autopsy	Absent left lung, left bronchus, and left pulmonary vessels; right lung 2-lobed with incomplete fissure; emphysema and interstitial pneumonia	No associated congenital anomalies elsewhere
de Toni 1934	4 mo. Female	At autopsy	Absent right lung; left lung hypertrophied; bronchopneumonia	Heart and mediastinum displaced to right
Wehr 1934	Newborn Female	At autopsy	Absent left lung and left pulmonary vessels; absent left bronchus; right bronchus had 1 eparterial and 2 hyparterial branches to right lung; 1 right pulmonary artery and 2 right pulmonary veins	Tracheoesophageal fistula; defective left diaphragm; vascular anomalies of heart; interventricular septal defect; spleen and stomach within left pleural cavity

TABLE 1.—*Cases of Aplasia of One Lung Classes One and Two (Schneider)—Continued*

Author and Year	Age and Sex	Time of Diagnosis	Lung Findings	Associated Findings
Rienhoff 1937	4½ mo. Male	At autopsy	Absent right lung and right bronchus; absent right pulmonary artery, except for nubbin-like primordium	Patent interventricular septum; imperforate anus
Bohnholtzer 1938	Newborn Unsexed	At autopsy	Absent right lung, right bronchus, and right pulmonary vessels; small left lung, completely lobed	Heart and mediastinum slightly shifted to right; riding aorta communicating with both ventricles; patent foramen ovale and ductus arteriosus; horseshoe kidneys; single dilated left ureter; internal and external genitalia lacking; no appendix; imperforate anus; sigmoid colon dilated
Jamuni and Ellis 1938	1 day Male Premature	At autopsy	Absent left lung; rudimentary left bronchus; fluid in left pleural cavity, lined by parietal pleura	Absent spleen; several accessory spleen-like structures; heart displaced to right
Wasmuht 1938	4 wk. Male	Suspected during life; established at autopsy	Absent right lung, right bronchus, and right pulmonary artery; trachea continued as left bronchus; left lung hypertrophied; incompletely lobed	Large thymus; heart shifted to right; patent ductus arteriosus and foramen ovale; large Meckel's diverticulum; chronic peritonitis and gastroenteritis
Wasmuht 1938	4 mo.	At autopsy; no clinical data	Absent right lung, right bronchus, and right pulmonary artery	Autopsy restricted to chest organs in situ; ductus arteriosus closed
Killingsworth and Hibbs 1939	72 hr. Female	At autopsy	Absent right lung, right bronchus, and right pulmonary vessels; left lung 1-lobed	Absent left kidney and ureter; only 1 ovary and fallopian tube
Heemstra 1939	6 wk. Male	During life; confirmed at autopsy	Absent right lung and right bronchus; absent right pulmonary artery; trachea led into left bronchus; left lung enlarged; partially lobed	Thorax symmetrical; right heart enlargement; shifted to right; patent ductus arteriosus
Saxl 1940	3 mo. Male	At autopsy	Absent right lung and right pulmonary vessels; nubbin-like right bronchus; left lung hypertrophied; accessory lobe at base	Heart shifted to right pleural cavity; absent right thumb and its metacarpal
Stokes and Brown 1940	12 yr. Female	During life at 4 mo. of age; still living	Absent left lung; remnant of left bronchus less than ¼ in. (0.64 cm.) in depth; right lung enlarged, portion herniating into left chest	Traction diverticulum of esophagus at level of 5th rib
Van Loon and Diamond 1941	3¼ yr. Female	During life; still living	Absent right lung and right main stem bronchus; absent right pulmonary vessels suggested by x-ray	No associated congenital anomalies reported
Geissler 1941	Stillborn Female	At autopsy	Absent right lung; right bronchus 3-4 mm. long; blind stump-like ending; normal left bronchus and branches; absent right pulmonary artery and veins; left lung hypertrophied	Absent true glottis; patent foramen ovale and ductus arteriosus; no deformities in other parts of body
Madigan 1941	23 yr. Female	During life; still living	Absent left lung; diminished caliber of left bronchial branches; right lung emphysematous and hypertrophied; trachea shifted to left	High site thoracic scoliosis; unable to establish air space in left pleural cavity on diagnostic pneumothorax
Castellanos and Pereiras 1942	10 yr. Female	During life; still living	Absent left lung; short left bronchus; single right pulmonary artery on angiocardiography	No associated congenital anomalies
Olcott and Dooley 1943	2 mo. Female	At autopsy	Absent right lung and right main stem bronchus; 4 mm. pocket at normal site of branching of right main stem bronchus; no tissue attached to its external aspect; left lung enlarged	Trachea formed an almost direct line with left bronchus; associated anomalies not mentioned
DeWeese and Howard 1944	6 mo. Male	During life; confirmed at autopsy	Absent right lung and right bronchus; left lung bronchopneumonia	Mediastinum shifted to right; heart somewhat dilated
Valle and Graham 1944	41 yr. Female	During life; confirmed by exploratory thoracotomy; still living at time of report	Absent left lung and left pulmonary vessels; short left bronchial stump; no lung tissue	Mediastinum displaced to left

TABLE 1.—*Cases of Aplasia of One Lung Classes One and Two (Schneider)—Continued*

Author and Year	Age and Sex	Time of Diagnosis	Lung Findings	Associated Findings
Valle and Graham 1944	5 yr. Male	During life at 9th month; still living; authors regard diagnosis as presumptive as thoracotomy not done	Absent left lung and left main bronchus	No associated congenital anomalies mentioned
Ferguson and Neuhauser 1944	4 mo. Female	During life; confirmed at autopsy	Absent right lung, right bronchus, and right pulmonary vessels; left lung with interstitial pneumonia	Patent ductus arteriosus; hypoplasia of spleen, kidneys, and liver; accessory spleen
Ferguson and Neuhauser 1944	8½ yr. Female	During life at 2 yr. 9 mo. of age; still living	Absent left lung; left main bronchus 1 cm. in length; right lung emphysematous; normal right bronchus	Heart and mediastinum shifted to left; absent left hand; temporary left facial palsy; asymmetrical chest cage
Ferguson and Neuhauser 1944	4 mo. Female	During life; still living	Absent right lung and right bronchus; trachea continued directly into left main bronchus; left lung emphysematous	Heart and mediastinum shifted to right; marked left dorsal scoliosis; some hemivertebrae; spina bifida; fused right 3d to 6th ribs
Ferguson and Neuhauser 1944	4 yr. Male	During life at 3d yr.; still living	Absent left lung and left main bronchus; right lung emphysematous; normal right bronchus	Heart and mediastinum shifted to left; harelip; cleft palate; asymmetrical chest cage
Lelong, Lemoyne, Charousset, and Lange 1944	26 mo. Female	During life at age of 11½ mo.; still living at time of report	Absent left lung and left bronchus	Heart and mediastinum slightly shifted to left; vascular anomalies of heart (details not given)
Cassubon, Cuculla, and Mosquera 1945	5½ mo. Female	At autopsy	"Aplastic" lung (left) 2 cm. by 1 cm.; absent left pulmonary vessels	Interauricular and interventricular septal defects
Garber 1945	8 yr. Female	During life at 1 yr. of age; followed for 7 yr.; still living	Absent left lung; small left main bronchus (1.5 cm.) with 2 small (2 mm.) branches; right lung moderately emphysematous	No associated congenital anomalies stated
Pierson 1946	5 mo. Female	During life; still living	Absent right lung and right bronchus; left lung hypertrophied; trachea continued into left main stem bronchus	No associated anomalies elsewhere
Smart 1946	30 yr. Female	During life; still living	Absent right lung and right bronchus	No associated anomalies stated
Smart 1946	29 yr. Female	During life; still living	Absent left lung; bronchoscopy unsuccessful on several attempts	Congenitally small trachea
Field 1946	1 yr. Female	During life; still living	Absent left lung; small left bronchus stump; diminished right middle lobe; rest of right lung enlarged; part herniated into anterior mediastinum; anomalous pectoral branch of right bronchus supplied right middle lobe	Microcephalus; talipes equinovarus; ptosis of eyelids; spina bifida of 1st and 2d thoracic levels
Widerman and Peters 1946	8 mo. Female	During life; still living	Absent right lung and right bronchus	Heart in right chest; 8th and 9th thoracic vertebrae fused
Burger 1947	2 mo. Female	At autopsy	Absent left lung, left bronchus, and left pulmonary vessels; trachea continued as right main bronchus; right lung bilobed; lacked eparterial bronchial branch and branch to apical lobe	No congenital anomalies elsewhere
Bowden 1947	15 mo. Female	At autopsy	Absent right lung and right pulmonary vessels; tiny right bronchial bud; left lung 2-lobed, cystic	No congenital anomalies elsewhere
Nesbit, Paul, and Middleton 1947	40 yr. Female	During life; still living	Absent left lung; 2.5 cm. stump of left main bronchus	No congenital anomalies elsewhere
Yount 1948	36 hr. 1 of twins	At autopsy	Absent right lung and right bronchus; trachea continued into left main bronchus; left lung 3-lobed	Dextrocardia; normal heart and mediastinum displaced into right chest; webbed neck; elevated scapulae
Yount 1948	6 mo. 1 of twins	During life; still living	Absent right lung	Deformed right ear lobe; absent right ear canal; skin tab on right cheek; dextrocardia

TABLE 2.—*Cases of Congenital Hypoplasia of One or Both Lungs*

Author and Year	Age and Sex	Time of Diagnosis	Lung Findings	Associated Findings
Förster 1860	Stillborn	At autopsy	Severely hypoplastic left lung lying on vertebral column; left bronchus and left pulmonary vessels very small; left pleural cavity filled with connective tissue	Microcephalus
Ratjen 1867	49 yr. Male	At autopsy	Left lung ¼ in. (0.64 cm.), 2-lobed, firm; contained chalk, fat, and cholesterol; left bronchus ended blindly 1 in. (2.54 cm.) beyond bifurcation; right lung hypertrophied; right bronchial branches larger	Right heart enlargement; Ratjen regarded as congenital lung defect; Schuchhardt as acquired because of lung pigment
Ponfick 1870	5 days Female	At autopsy	Right lung small, 5 by 3.5 by 2.5 cm., airless fetal alveolar structures and thick-walled vessels; trachea and bronchi and vessels at hilus normal; left lung large	Mediastinum shifted to right; right heart enlarged; interauricular and interventricular septal defects; patent ductus arteriosus; coarctation of right pulmonary artery at area of ductus arteriosus; hydroperieardium
Tyson 1874	38 yr. Female	At autopsy	Hypoplastic left lung ⅓ normal size; upper lobe 4½ in. by 3½ in. (11.4 cm. by 8.8 cm.), lower lobe 3¼ in. by 2¼ in. (8.3 cm. by 5.7 cm.)	Heart hypertrophied; bilateral hydrothorax; bronchi and pulmonary vessels not described
Klebs 1874	Newborn	At autopsy	Left lung anlage, size of hazelnut at end of esophageal diverticulum; no lung tissue in left pleural cavity, reduced in size; left bronchus ended blindly; right lung 2-lobed	Tracheoesophageal fistula
Miller 1894-1895	4 wk. Male	At autopsy	Pea-sized left lung attached to a thin bronchus; pneumonia of right lung	Associated anomalies elsewhere and pulmonary vessels not mentioned
Miller 1894-1895	6 wk. Male	At autopsy	Pea-sized left lung attached to a narrow bronchus	Associated anomalies elsewhere and pulmonary vessels not mentioned
Miller 1894-1895	2 days Female	At autopsy	2 cm.-sized left lung attached to narrow bronchus	Associated anomalies elsewhere and pulmonary vessels not mentioned
Sieveking 1895	56 yr. Male	At autopsy	Extremely hypoplastic left lung, partly fibrous, attached to chest wall, fetal structure; 2 lobes; right lung hypertrophied and emphysematous, crossing in part the mediastinum; bronchi and pulmonary vessels not described	Heart occupied left pleural cavity; no other congenital anomalies; chronic syphilis; amyloidosis of liver, spleen, kidneys, adrenals, and intestines; author bases diagnosis of congenital lung condition on lack of functioning left lung tissue, shift of heart to left, hypertrophic right lung, and slight narrowing of left hemithorax
Eppinger and Schauenstein 1902 Case 2	4 mo. Male	At autopsy	Right lung small; absent 1 lobe; in dorsal half of right thoracic cavity; left lung enlarged and shifted to right; right pulmonary artery and right bronchus small; microscopic examination of right lung showed fetal lung tissue	Heart shifted to right
Eppinger and Schauenstein 1902 Case 1	Newborn Female	At autopsy	Both lungs severely hypoplastic: right 3 cm. by 1.3 cm. by 5.5 cm., left 1.3 cm. by 1.5 cm. by 1.3 cm., corresponding to child at half the gestation period; microscopic examination showed small open alveoli, flattened epithelium, much elastic connective tissue; normal trachea, bronchi, and pulmonary vessels	Enlarged unaltered heart; large thymus; no pleural adhesions; marked bilateral hydrothorax
von Graff 1905	2½ days Male	At autopsy	Hypoplastic right lung; 20 mm. by 16 mm. by 4 mm.; fetal lung tissue; vestigial right pulmonary artery; retromediastinal right bronchus; absent right pulmonary veins; left lung hypertrophied; 2-lobed; no pleural adhesions	Heart and mediastinum shifted to right; absent right phrenic nerve; slit-like foramen ovale; patent ductus arteriosus; atresia ani serotalis; large thymus
Zipkin 1907	Stillborn Female	At autopsy	Small right lung; 3-lobed; 5 cm. by 2.5 cm. by 1.2 cm.; 6 gm.; fetal tissue; left lung enlarged; rhabdomyoma and pneumonia	No anomalies elsewhere; hydrops of entire body

TABLE 2.—*Cases of Congenital Hypoplasia of One or Both Lungs—Continued*

Author and Year	Age and Sex	Time of Diagnosis	Lung Findings	Associated Findings
Berliner 1909	2 days Female	At autopsy	Absent right upper and middle lobes with corresponding bronchial branches ending blindly; small right lower lobe 10 cm. by 6 cm.; left lung enlarged; both pulmonary arteries present	Adhesive pleuritis of right side; heart and mediastinum shifted to right
Elward 1936	43 yr. Male	At autopsy	Left lung fibrotic, rudimentary, 3 in. by 1½ in. (7.6 cm. by 3.8 cm.); attached to posterior chest wall; left bronchus narrowed on bronchoscopy; enclosed in adhesions at autopsy; right lung hypertrophied; left pulmonary artery and branches markedly diminished in size	No other anomalies
Lüdin and Werthemann 1936	50 yr. Female	At autopsy	Hypoplastic thin left lung 12 mm. in diameter and 2 mm. in thickness; rudimentary lung tissue; left main bronchus stenosed by chondromatous body within wall; left upper lobe bronchus absent; right lung enlarged; 5 lobes; vestigial left pulmonary artery; normal right pulmonary artery	Persistent sinus venosus communicating with inferior vena cava; persistent left superior vena cava; union of gallbladder with duodenum
Paul 1928	2¼ yr. Female	At autopsy	Left lung hypoplastic, 3 cm. by 2 cm. by 1 cm. fetal structure; left main bronchus ended blindly after 1 cm.; right pulmonary artery continued as sole branch of main pulmonary artery; rudimentary left pulmonary artery from thoracic aorta; right lung enlarged and 2-lobed	Small bronchial communication from left lung to lower esophagus; right heart enlarged; hydropericardium; patent ductus arteriosus
Altmann 1929 Case 1	Stillborn Female	At autopsy	Left lung unlobed, rudimentary, 20 mm. by 12 mm. by 6 mm. behind heart; left bronchus much narrowed; absent left pulmonary artery; that lung supplied by small branches from descending aorta; right lung normal except for atelectasis; absent left pulmonary veins	Heart shifted to left; patent foramen ovale; patent ductus arteriosus; absent left first phalanx and metacarpals; bilateral club feet; small liver; bilateral polycystic kidneys; right larger than left; bilateral absent upper parts of ureters; absent vagina in lower third; absent appendix
Altmann 1929 Case 2	Newborn Female	At autopsy	Left lung half size of right lung; trachea and bronchi normal; right lung enlarged; microscopic examination showed both lungs normal	No other anomalies
Botar and Orta Llorca 1932	Newborn	At autopsy	Rudimentary right lung 4 cm. by 2.4 cm. corresponding to right lower lobe, lying in right diaphragmatic gutter and covered by heart; trachea curved into left lung and giving out normal left bronchi; right bronchus very small, near midline, and had only one branch; left lung normal; small right pulmonary artery; many complicated small branches	Dextrocardia with enlargement and interventricular septal defect; large thymus; pulmonary veins emptied into azygous vein; left superior vena cava persisted; embryonic structures of kidney, ureters, and uterus; atrophy of small intestines, rectum, and anus; absent omentum; large spleen and small liver; accessory spleens and livers; large Meckel's diverticulum
Formijne 1938	19 yr. Female	During life; confirmed at autopsy	Nubbin-sized severely hypoplastic left lung at end of 2 small twig-like left bronchial branches ending blindly; rudimentary lung on microscopic study; right lung hypertrophied; very small pulmonary arteries and veins	Thorax asymmetrical; 2d right rib split at neck; periostitis of left midfemur; hypertrophic osteoarthropathy
Yampolsky and Fowler 1938	2½ yr. Male Died 2 hr. after admission	At autopsy	Right lung vestigial, 12 gm.; right bronchus had congenital stricture, 1 mm. lumen, at 1 cm. below carina; 1-lobed left lung, 160 gm.	Heart displaced to right
Cholessier and Bloedorn 1939	43 yr. Male	At autopsy	Left lung remnant 4.5 cm. by 3 cm. by 1.5 cm. fibrotic; microscopically, fetal epithelium of alveoli; small left bronchus and small left pulmonary artery; right lung hypertrophied; adhesions in both pleural cavities	Enlarged heart, displaced into left pleural cavity; right ventricular hypertrophy; middle pulmonary cusp fenestrated; malformed left auricular appendage

TABLE 2.—*Cases of Congenital Hypoplasia of One or Both Lungs—Continued*

Author and Year	Age and Sex	Time of Diagnosis	Lung Findings	Associated Findings
Purriel and Piaggio 1939	22 yr. Male	During life; still living	Absent right lung; right bronchus ends abruptly after dilatation (bronchogram); normal right lung; right intercostal spaces narrower than left; asymmetry of thorax	Pulmonary vessels not investigated; dextrocardia; mediastinum shifted to right; strabismus
Saxl 1940	14 days Female	At autopsy	Hypoplastic right lung; left lung hypertrophied and emphysematous	Dextrocardia
Saxl 1940	4 mo.	At autopsy	Hypoplastic left lung; right lung bronchopneumonia; no description of great vessels	Duplication of left first and second phalanges
Castellanos and Pereiras 1942	10 yr. Female	During life; still living at time of report	Absent right upper lobe bronchus and corresponding lung lobe; hypoplastic right bronchial branches; right pulmonary artery present	No associated congenital anomalies described
Castellanos and Pereiras 1942	1 day	During life; died after 21 days	Severely hypoplastic left lung; 2 cm. by 3 cm.; bronchi and pulmonary vessels not described	Tetralogy type of cardiac anomaly (single ventricle, interauricular septal defect), meningocele, imperforate anus, persistent left superior vena cava
Gartside 1943	7 yr. Male	During life; still living at time of report	Absent upper and middle portions of left lung; wedge-shaped lower left lung segment above diaphragm in costovertebral gutter; left bronchus half normal length, gave off many slender twigs; partial rotation of right lung	Spina bifida, wedge-shaped vertebrae, cervical ribs, fusion of ribs, congenital high scapula, and congenital heart (probably a septal defect)
Ferguson and Neuhauser 1944	6 mo. Male	During life; still living at time of report	Absence of most of left lung; left main bronchus 3 cm. in length and gave off 2 narrow first subdivisions; heart and mediastinum shifted to left	Malformed left external ear
Field 1946	10 yr. Female	During life; still living at time of report	No left lung seen on x-ray; left bronchus had only a single lower lobe small branch; enlarged right lung and right bronchus	No associated congenital anomalies elsewhere stated
Field 1946	5½ mo. Female	During life; confirmed at autopsy	Right lung hypoplastic with fetal structure on sections; small right bronchus; left main bronchus enlarged	Patent interventricular septum; heart enlarged
Field 1946	2½ yr. Female	During life; still living at time of report	Right lung had small right middle and lower lobe bronchi and corresponding lung structures; large right upper lobe bronchus left lung and bronchus normal	Trachea deviated to the right
Field 1946	6 yr. Female	During life; still living at time of report	Hypoplastic left lung displaced backward; absent left lingular bronchus; right lung crossed the midline; trachea shifted to left	Cleft palate, stenosis of trachea, congenital deformities of cervical vertebrae, and torticollis
Fontan, Verger, Mouliès, and Martin 1947	20 days	At autopsy	Absent right upper and middle lobes; small single-lobed right lung corresponding to right lower lobe lay behind heart in right pleural cavity; received 3 small bronchi; left lung enlarged and 2-lobed; left bronchus more developed than right; pneumonia of right lung	Dextrocardia; normally placed abdominal viscera; no anomalies in great vessels
Naclerio and Hochberg 1950	5½ mo.	During life; confirmed at autopsy	Absent right upper lobe, middle lobe, and corresponding bronchi	Heart and mediastinum shifted to right; tracheoesophageal fistula with esophageal atresia; horseshoe kidneys; fused lower lumbar vertebrae
Naclerio and Hochberg 1950	13 yr. Male	During life; confirmed at exploratory thoracotomy; still living	Absent right upper and middle lung lobes and corresponding bronchi; right lower lobe emphysematous in superior segment and indurated in basilar segment; normal left lung and bronchus and corresponding pulmonary vessels	Trachea and mediastinum shifted to right; no congenital anomalies elsewhere
Naclerio and Hochberg 1950	7 yr. Male	During life; still living	Absent right upper and middle lobes and their bronchi; left lung emphysematous	Mediastinum and trachea shifted to right

alveoli and of partial atelectasis. The alveolar contents of the latter areas varied from a fibrinous exudate with polymorphonuclear leucocytes and a few eosinophiles to edema fluid with phagocytic macrophages having ingested yellow-brown material or foamy substance in the cytoplasm. Where the alveoli were collapsed, the interalveolar septums were thickened. The bronchioles within the atelectatic area were lined by a simple columnar epithelium, which was infolded and infiltrated in the lamina propria by polymorphonuclear leucocytes. These changes in the left lung were interpreted as an acute bronchopneumonia, partial atelectasis, and acute passive congestion with retention of lipiodol material and with acute bronchitis and bronchiolitis.

Due to autopsy restriction, examination of the brain and investigation of the skeletal system for the anomalies noted on the x-ray examination could not be made.

COMMENT

Schneider divided these malformations into three classes. Class 1 consists of cases in which no trace of a bronchus or lung is found; in Class 2, there is no lung tissue, but some trace of a bronchus is found; and in Class 3, the main bronchus is incompletely developed, and there is marked hypoplasia of one or both lungs.

TABLE 3.—*Cases of Congenital Absence of Both Lungs*

Author and Year	Age and Sex	Time of Diagnosis	Lung Findings	Associated Findings
Roderer 1754	Fetus	At autopsy	Thoracic cavity contained only thymus and incompletely developed heart; in place of lungs a thick jelly-like watery tissue without trace of pulmonary arteries and bronchi	Larynx ended blindly
Schmit 1893	Newborn Premature	At autopsy	Both lungs absent; absent pulmonary veins; trachea not separated from esophageal wall; pulmonary artery connected as large ductus botalli with descending aorta	Only heart in thoracic cavity; no pleural membranes; high diaphragm; larynx well developed
Saito 1902	Fetus	At autopsy	Both lungs absent; trachea separated from esophagus; larynx ended blindly at level of cricoid cartilage	Only heart in thoracic cavity; isthmus of thyroid lacking
Allen and Affelbach 1925	Newborn Female Lived 20 min.	At autopsy	Both lungs absent; trachea ended blindly at level of root of heart with 2 1-mm. pouches on each side; no vestigial lung tissue	All other organs normal; esophagus intact

Our case presents a true agenesis with no trace of the lung, bronchus, or vascular supply on the right side. Since it is sometimes impossible to differentiate clearly Class 1 from Class 2, even from the autopsy findings, we placed both classes under one category. Seventy-three cases, the patients varying in age from stillborn and newborn infants to a 72-year-old woman, were reviewed (Table 1). The diagnosis was made during life in 25 instances. There are 38 cases of hypoplasia of one or both lungs, 13 of which were diagnosed during life (Table 2). The cases of 4 newborn infants with congenital absence of both lungs were found in the literature (Table 3), while 21 patients were found with either agenesis or congenitally hypoplastic lung, the description being either incomplete or the condition not definitely established by special clinical methods or by postmortem examination (Table 4).

A clinical diagnosis for these patients can usually be strongly suspected on the basis of the physical findings, the x-ray and fluoroscopic findings of the displacement of the mediastinum and of the heart toward the affected side, especially if associated skeletal anomalies of the spine and of the ribs and other anomalies are also present. Bronchoscopic examination, with bronchography and angiocardiog-

TABLE 4.—*Cases of Agenetic or Congenitally Hypoplastic Lung with Incomplete Descriptions or Not Established Beyond Doubt by Special Clinic Methods or by Postmortem Confirmation*

Author and Year	Age and Sex	Time of Diagnosis	Lung Findings	Associated Findings
Riviere 1779	Young male	At autopsy	Absent right lung; trachea and bronchi not described	Comment: probably aplasia of right lung
de Pozzi 1873	Adult Female	At autopsy	Absent left lung; trachea and bronchi not described	Abnormally short intestinal tract; enlarged spleen; comment: probable aplasia of left lung
Sömmering 1820	Child	At autopsy	Absent right lung; trachea and bronchi not described	Comment: probable aplasia or severe hypoplasia of right lung
Meckel 1794	Newborn	At autopsy	Defect of left lung; right lung 2-lobed; trachea, bronchi, and pulmonary vessels not described	Cleft palate and cranial defect; comment: inconclusive
Klebs 1874 Case 1	Not stated	At autopsy	Cartilaginous bronchus	Comment: O. Meyer, 1910, places this case in Schneider 2 group
Klebs 1874 Case 2	Not stated	At autopsy	Absent lung with bronchial rudiment; not stated which lung was involved	Comment: O. Meyer places this case in Schneider 2 group
Coats 1883	40 yr. Male	At autopsy	Left lung small, no lung tissue; only fibrous tissue and dilated bronchi; anthracotic pigment; right lung enlarged, not emphysematous; left pulmonary artery ⅓ as wide as right pulmonary artery; both main bronchi equally large	Presence of pigment and presence of left bronchus unreduced make it a likely acquired lesion
Schlicht 1885	Adult Female	At autopsy	Left lung very small with bronchiectasis; right lung large and also occupied much of left pleural cavity; pneumonia	Thorax full and normally formed; regarded by Schneider as early extrauterine atrophy of left lung
Recklinghausen 1885	40 yr. Male	At autopsy	Left lung up to ½ a man's fist in size; atelectatic without pigment; right lung enlarged, emphysematous, extending slightly into left chest; left lung firmly attached to chest wall	Trachea, bronchi, and pulmonary vessels not described
Schuchhardt 1885	28 yr. Male	At autopsy	Left lung reduced to 14 cm. by 8 cm., 2-lobed; alveoli, lined by large epithelial cells, alveolar vessels thick walled and with increased musculature; richly pigmented; enlarged right lung, partly extending into left pleural cavity; all bronchi and branches present; left bronchus thickened and narrowed; pulmonary vessels apparently intact; ductus arteriosus closed	Enlarged right heart; author regards as high-grade hypoplasia after an atelectasis and not congenital
Wollmann 1891	24 yr. Female	At autopsy	Very small left lung with extensive bronchiectasis without trace of lung tissue or lung pigment; no cartilage in bronchi; right lung hypertrophic; left lung firmly bound at apex and posterior to thoracic vertebrae and to diaphragm	Slight scoliosis; comment: despite lack of pigment an acquired condition is not ruled out; Schneider considered as atelectatic bronchiectasis
Reinhold 1893	56 yr. Female	At autopsy	Small left lung without alteration of illness or malformation; right lung hypertrophied; no microscopic investigation	Heart shifted to left; comment: inconclusive
Neisser 1900	42 yr. Male	During life; still living at time of report	Small left lung of thickened tissue and enormously enlarged right lung on x-ray	Heart displaced to left; no chest asymmetry; slight left-sided scoliosis with elevation of right shoulder
Thorel 1903	22 yr. Female	At autopsy	Marked hypoplasia of upper lobe of right lung; calcified material, firm adhesions; bronchial branches narrowed	Tuberculosis not established; comment: probably an acquired lesion
Meyer 1910	27 yr. Male	During life; still living at time of report	Absent left lung on x-ray; chest symmetrical; no bronchoscopy or bronchograms	Heart shifted to left; comment: suggestive of aplastic left lung
Osterroht 1910	18½ yr. Male	During life; still living at time of report	Absent left lung on x-ray; chest symmetrical; no bronchoscopy or bronchograms	Heart shifted to left; comment: suggestive of hypoplasia or agenesis of left lung
Finkelstein 1924	Young child One of identical twins Female	At autopsy	Absent left lung; bronchi and pulmonary vessels not described	Comment: probable aplasia of left lung
Finkelstein 1924	Young child One of identical twins Female	At autopsy	Absent right lung; bronchi and pulmonary vessels not described; compare with preceding case and with cases observed by Yount, 1948	Comment: probable aplasia of right lung (opposite lung from preceding case)
Pruys, Veen and Zuidema 1939	25 yr. Male	During life; still living at time of report	Absent left lung tissue on chest x-ray; mediastinum not displaced to affected side; bronchograms showed saccular bronchiectasis	No asymmetry of thorax
Toro and Sa 1944	Not given	Unknown	Agenesis; details not given	Comment: of 21 cases of congenital lung anomalies in routine chest x-rays, there were 3 cases of pulmonary agenesis

raphy, may be necessary in some cases to establish the diagnosis. However, because of the danger connected with these procedures, the latter should be performed only after due clinical consideration of each individual case, and then only by experienced bronchoscopists.

CONCLUSION

A case of agenesis of the right lung, Schneider Class 1, in a 2-day-old girl is reported. A complete review of previously-reported cases is presented.

BIBLIOGRAPHY

APLASIA OF ONE LUNG

Bell, J., and Bell, C.: The Anatomy and Physiology of the Human Body, Ed. 1, London, Longman, Hurst & Co., 1823, p. 74; Ed. 5, N. Y., Collins & Co., 1827, p. 416; Ed. 7, 1829, p. 69.

Bohnholtzer, E.: Beitrag zur Kenntnis einseitiger Lungenagenesien, Beitr. path. Anat. **101**:614, 1938.

Bönniger, M.: Angeborener Defekt der rechten Lunge, Med. Klin. **24**:258, 1928; cited by Bohnholtzer, E.: Beitrag zur Kenntnis einseitiger Lungenagenesien, Beitr. path. Anat. **101**:614, 1938.

Ein weiterer Fall von angeborenem Defekt der rechten Lunge kompliziert durch rechtsseitigen Pneumothorax, Med. Klin. **27**:1568, 1931.

Bowden, K. M.: Absence of Right Lung: Congenital Cystic Malformation of the Left Lung, M. J. Australia **1**:646, 1947.

Burger, R. A.: Agenesis of the Lung, with a Review of the Literature, Am. J. Dis. Child. **73**:481, 1947.

Casaubon, A.; Cucullu, L. M., and Mosquera, J. E.: Aplasia del pulmón izquierdo y malformación cardíaca, Arch. argent. pediat. **24**:3, 1945; cited by Burger, R. A.: Agenesis of Lung with Review of Literature, Am. J. Dis. Child. **73**:481, 1947.

Castellanos, A., and Pereiras, R.: Pulmonary Agenesia: Report of 3 Cases, and General Review, Biol. Soc. cubana pediat. **14**:268, 1942.

Dannheisser, F.: Ein Fall von Totaldefekt der linken Lunge, Beitr. path. Anat. **76**:87, 1926.

de Toni, C.: Sopra un caso di agenesia del polmone destro con destrocardia, Clin. pediat. **16**:241, 1934; cited by Hurwitz, S., and Stephens, H. B.: Agenesis of Lung: Review of Literature and Report of Case, Am. J. M. Sc. **193**:81, 1937.

DeWeese, E. R., and Howard, J. C., Jr.: Congenital Absence of a Lung Diagnosed Before Death, Radiology **42**:389, 1944.

Dyson, J. E.: Congenital Absence of Lung, J. Iowa M. Soc. **24**:442, 1934.

Ellis, A. G.: Congenital Absence of Lung, Am. J. M. Sc. **154**:33, 1917.

Ferguson, C. F., and Neuhauser, E. B. D.: Congenital Absence of the Lung (Agenesis) and Other Anomalies of the Tracheobronchial Tree, Am. J. Roentgenol. **52**:459, 1944.

Field, C. E.: Pulmonary Agenesis and Hypoplasia, Arch. Dis. Childhood **21**:61, 1946.

Findlayson, A.: Complete Absence of the Right Lung, Bronchus, and Pulmonary Vessels in a Recently Born Child, Proceedings of the Anatomical Society of Great Britain and Ireland, 1902, p. 38.

Finner, L. L.: Congenital Absence of One Lung: Report of a Case with Autopsy, Clifton M. Bull. **18**:35, 1932.

Garber, R. L.: Congenital Aplasia of the Lung, Am. J. Roentgenol. **53**:129, 1945.

Geissler, J.: Zur Kenntnis der einseitigen Lungenagenesie, Zentralbl. allg. Path. **77**:339, 1941.

Gilkey, H. M.: Congenital Absence of the Lung: Report of Case, J. Missouri M. A. **25**:296, 1928.

Goehle, O. L.: Foreign Body in the Bronchus of a Child with Congenital Absence of the Other Lung, Am. J. Dis. Child. **35**:733, 1928.

Gross, W.: Ein Fall von Agenesie der linken Lunge, Beitr. path. Anat. **36**:487, 1905; cited by Ellis, A. G.: Congenital Absence of Lung, Am. J. M. Sc. **154**:33, 1917.

Gruber, W.: Mangel der rechten Lunge, Oesterr. Ztschr. prakt. Heilk. **16**:7, 1870; cited by Fürst, L.: Missbildungen der Lunge, in Handbuch der Kinderkrankheiten, edited by C. Gerhardt, Vol. 3, Pt. 2, Tübingen, H. Laupp, 1878, p. 553.

Mangel der linken Lunge bei Vorkommen ein Nebenleber und dreier Nebenmilzen: 2. Fall eigener Beobachtung des Mangels einer der Lungen, Arch. path. Anat. **102**:11, 1885; cited by Schneider, P., in Schwalbe, E.: Morphologie der Missbildungen des Menschen und der Tiere, Jena, G. Fischer, 1909-1913, Vol. 3, Pt. 2, p. 772.

Haberlein, C., and Klinz: Abhandlungen der K. K. Josephischen Mediz Chirurg., Akademie zu Wien. **1**:271, 1787, cited by Fürst, L.: Missbildungen der Lunge, in Handbuch der Kinderkrankheiten, edited by C. Gerhardt, Vol. 3, Pt. 2, Tübingen, H. Laupp, 1878, p. 553; cited by Schneider, P., in Schwalbe, E,: Die Morphologie der Missbildungen des Menschen und Tiere, Jena, G. Fischer, 1909-1913, Vol. 3, Pt. 2, p. 772; cited by Meckel, J. F.: Handbuch der pathologischen Anatomie, Leipzig, C. H. Reclam, 1812-1816, Vol. 1.

Haberlein, C.: Rev. mens. mal. l'enfance **2**:554, 1884; cited by Ellis, A. G.: Congenital Absence of Lung, Am. J. M. Sc. **154**:33, 1917.

Hanson, R.: Babe with One Lung, J. A. M. A. **37**:701, 1901.

Heemstra, T.: Case of Agenesia of the Right Lung, Nederl. tijdschr. geneesk. **83**:2750, 1939.

Heerup, L.: Case of Absence of Left Lung, Hospitalstid. **70**:1165, 1927; cited by Hurwitz, S., and Stephens, H. B.: Agenesis of Lung: Review of Literature and Report of Case, Am. J. M. Sc. **193**:81, 1937.

Hein: Fehlen der rechten Lunge: Blausucht, Wchnschr. ges. Heilk. **33**:536, 1837; cited by Ellis, A. G.: Congenital Absence of Lung, Am. J. M. Sc. **154**:33, 1917.

Hepner, F.: Agenesie der linken Lunge, Arch. Kinderh. **103**:92, 1934; cited by Hurwitz, S., and Stephens, H. B.: Agenesis of Lung: Review of Literature and Report of Case, Am. J. M. Sc. **193**:81, 1937.

Herrero, F.: Caso notable de agenesia de un pulmon, Corresp. méd., Madrid **9**:372, 1874; cited by Ellis, A. G.: Congenital Absence of Lung, Am. J. Med. Sc. **154**:33, 1917.

Heyfelder: Amtliche Bericht über die Versammlung der Naturforscher und Ärzte zu Stuttgart im Sept. 1834, Stuttgart, 1835, p. 107; cited by Fürst, L.: Missbildungen der Lunge, in Handbuch der Kinderkrankheiten, edited by C. Gerhardt, Vol. 3, Pt. 2, Tübingen, H. Laupp, 1878, p. 553; cited by Bohnholtzer, E.: Beitrag zur Kenntnis einseitiger Lungenagenesien, Beitr. path. Anat. **101**:614, 1938.

Jamuni, A., and Ellis, A .G.: Note on Case of Congenital Absence of Left Lung, Am. J. M. Sc. **196**:824, 1938.

Jarisch, A.: Fehlen einer Lunge bei einem Frontsoldaten, Wien. Klin. Wchnschr. **32**:736, 1919; cited by Hurwitz, S., and Stephens, H. B.: Agenesis of Lung: Review of Literature and Report of Case, Am. J. M. Sc. **193**:81, 1937.

Killingsworth, W. P., and Hibbs, W. G.: Agenesis of Lung: Review of Literature and Report of Case, Am. J. Dis. Child. **58**:571, 1939.

Knott, A.: Angeborener vollständiger Mangel der rechten Lunge, Ztschr. Kinderh. **56**:338, 1934; cited by Hurwitz, S. and Stephens, H. B.: Agenesis of Lung: Review of Literature and Report of Case, Am. J. M. Sc. **193**:81, 1937.

Lelong, M.; Lemoyne, J.; Charousset, and Lange: Un cas d'absence congénitale due poumon gauche, Arch. franç. de pédiat. **2**:31, 1944.

Levy, C. S.: Congenital Absence of One Lung, Am. J. M. Sc. **159**:237, 1920.

Lochow: Bericht über die Sitzung der russischen pathologischen Gesellschaft (Abt. Leningrad) 1928, Ztschr. Path. 464 Nr. VI, 170; cited by Bohnholtzer, E.: Beitrag zur Kenntnis einseitiger Lungenagenesien, Beitr. path. Anat. **101**:614, 1938.

Madigan, D. G.: Case for Discussion, with Special Allusion to Congenital Absence of the Lung, Tubercle **22**:144, 1941.

Maschka: Seltene Missbildung bei einem neugeborenen Kinde, Allg. Wien. med. Ztschr. **7**:78, 1862; cited by Fürst, L.: Missbildungen der Lunge, in Handbuch der Kinderkrankheiten, edited by C. Gerhardt, Vol. 3, Pt. 2, Tübingen, H. Laupp, 1878, p. 553.

Minne, J., and Gernez, L.: Agénésie complète du poumon gauche chez un nouveau-né exencéphale, Ann. anat. path. **10**:503, 1933; cited by Hurwitz, S., and Stephens, H. B.: Agenesis of Lung: Review of Literature and Report of Case, Am. J. M. Sc. **193**:81, 1937.

Mitchell, H. E.: Unusual Cases of Foreign Bodies in the Food and Air Passages, Ohio M. J. **32**:950, 1936.

Agenesis of Right Lung with Death Following Aspiration of Foreign Bodies into Left Lung, Ann. Otol. Rhin. & Laryng. **55**:609, 1946.

Muhamed, K. S. N.: Absence of Left Lung, Indian M. Gaz. **58**:262, 1923.

Nesbit, W. M.; Paul, L. W., and Middleton, W. S.: Congenital Aplasia of the Lung: Case Report, Am. J. Roentgenol. **57**:446, 1947.

Oberwarth, E.: Uber angeborene Agenesie einer Lunge, Jahrb. Kinderh. **60**:383, 1904; cited by Bohnholtzer, E.: Beitrag zur Kenntnis einseitiger Lungenagenesien, Beitr. path. Anat. **101**: 614, 1938.

Olcott, C. T., and Dooley, S. W.: Agenesis of Lung in an Infant, Am. J. Dis. Child. **65**:776, 1943.

Pierson, W. M.: Aplasia of the Lung, Ann. Otol. Rhin. & Laryng. **55**:604, 1946.

Rienhoff, W. F., Jr.: Intrathoracic Anatomical Readjustments Following Complete Ablation of One Lung, J. Thoracic Surg. **6**:254, 1937.

Saxl, O.: Beitrag zur Diagnose der Lungenagenesie, Ann. pædiat. **154**:180, 1940.

Smart, J.: Complete Congenital Agenesis of a Lung, Quart. J. Med. **15**:125, 1946.

Stein, cited by Fürst, in Gerhardt's Handbuch der Kinderkrankheiten 3 **II**:553, 1878.

Stokes, H. L., and Brown, C. J. O.: Agenesis of the Lung: Report of a Case, M. J. Australia **1**:49, 1940.

Tebbutt, A. H.: Congenital Absence of Left Lung, M. J. Australia **2**:430, 1918; cited by Hurwitz, S., and Stephens, H. B.: Agenesis of Lung: Review of Literature and Report of Case, Am. J. M. Sc. **193**:81, 1937.

Théremin, E.: Deux cas d'absence congénital du poumon gauche, Rev. mens. mal. enfance **2**:554, 1884; cited by P. Schneider in Schwalbe, E.: Die Morphologie der Missbildungen des Menschen und der Tiere, Jena, G. Fischer, 1909-1913, Vol. 3, Pt. 2, p. 772.

Tichomiroff, M.: Ein Fall von congenitalem Mangel der linken Lunge mit Persistenz der linken oberen Hohlvene bei einem erwachsenen Menschen, Internat. Montasschr. Anat. u. Physiol. **12**:24, 1895; cited by Ellis, A. G.: Congenital Absence of Lung, Am. J. M. Sc. **154**:33, 1917.

Valle, A. R., and Graham, E. A.: Agenesis of the Lung, J. Thoracic Surg. **13**:345, 1944.

Van Loon, E. L., and Diamond, S.: Congenital Absence of Right Lung: Its Occurrence in Healthy Child, Am. J. Dis. Child. **62**:584, 1941.

von Eiken C.: Med. Klin. **47**:1836, 1907; cited by Bohnholtzer, E.: Beitrag zur Kenntnis einseitiger Lungenagenesien, Beitr. path. Anat. **101**:614, 1938.

Wasmuht, K.: Über zwei Fälle von Hyperplasie der linken und totaler Agenesie der rechten Lunge, Frankfurt. Ztschr. Path. **52**:519, 1938.

Wehr, C.: Inaugeraldissertation, Göttingen, 1934; cited by Bohnholtzer, E.: Beitrag zur Kenntnis einseitiger Lungenagenesien, Beitr. path. Anat. **101**:614, 1938.

Welsch, K.: Ein Fall von Mangel der rechten Lunge, Frankfurt. Ztschr. Path. **36**:192, 1928.

Widerman, A., and Peters, C. H.: Agenesis of the Lung: Case Report, J. Pediat. **28**:204, 1946.

Yount, F.: Agenesis of Right Lung in Each of Identical Twins, Arizona Med. **5**:48, 1948.

CONGENITAL HYPOPLASIA OF ONE OR BOTH LUNGS

Altmann, F.: Zur Kenntnis der Lungenhypoplasien, Ztschr. ges. Anat. **88**:500, 1928.

Berliner, M.: Ein Fall von Agenesie des Ober- und Mittellappens der rechten Lunge mit Dextroversio cordis, Wien. med. Wchnschr. **59**:1424, 1909.

Botár, J., and Orts Llorca, F.: Les Anomalies du poumon par défaut: Absence partielle due poumon droit chez un nouveau-né humain, Ann. anat. path. **9**:820, 1932.

Castellanos, A., and Pereiras, R.: Pulmonary Agenesia: Report of 3 Cases, and General Review, Bol. Soc. cubana pediat. **14**:268, 1942.

Choisser, R. M., and Bloedorn, W. A.: Agenesis of the Lung, Internat. Clin. **2**:243, 1939.

Elward, J. F.: Roentgen Aspects of Congenital Aplasia of Lung, Radiology **27**:667, 1936.

Eppinger, H., and Schauenstein, W.: Krankheiten der Lungen: Angeborene Krankheiten, in Lubarsch—Ostertag's Ergebnisse der allgemeinen Pathologie und pathologischen Anatomie Vol. 8 (Part 1): (7) 267-285 (1902). 2 Cases.

Ferguson, C. F., and Neuhauser, E. B. D.: Congenital Absence of the Lung (Agenesis) and Other Anomalies of the Tracheobronchial Tree, Am. J. Roentgenol. **52**:459, 1944.

Field, C. E.: Pulmonary Agenesis and Hypoplasia, Arch. Dis. Childhood **21**:61, 1946.

Fontan, A.; Verger, P.; Moulies, A., and Martin, C.: Dextrocardie congénitale par agénésie des deux lobes supérieur et moyen droit du poumon droit: Survie vingt jours, J. méd. Bordeaux **124**:126, 1947.

Formijne, P.: Agenesia and Hypoplasia of the Lungs, Nederl. tijdschr. geneesk. **82**:5482, 1938.

Förster, A.: Verh. d. phys.-med. Gesellsch. in Wurzburg, **10**:10, 1860; cited by Fürst, L.: Missbildungen der Lunge, in Handbuch der Kinderkrankheiten, edited by C. Gerhardt, Vol. 3, Pt. 2, Tübingen, H. Laupp, 1878, p. 553.

Die Missbildung des Menschen systematisch dargestellt, Ed. 2, Jena, F. Mouke, 1865, p. 106; cited by Schuchhardt, K.: Hochgradige Atrophie (inveterirte Atelectase) der linken Lunge mit compensatorischer Hypertrophie der rechten, Arch. path. anat. **101**:71, 1885.

Gartside, V. O. B.: Agenesis of the Lung: Report of a Case, Brit. J. Radiol. **16**:69, 1943.

Klebs, E.: Missbildungen der Lunge, Ärztl. Korrespondenzbl. Böhmen **2**:111, 1874; cited by Meyer, O.: Über einseitige congenitale Lungen atrophie, Arch. Entwcklngs.-mechn. Organ. **30**:342, 1910.

Lüdin, M., and Werthemann, A.: Ein Fall von angeborenem Mangel der linken Lunge, Fortschr. Geb. Röntgenstrahlen **53**:273, 1936.

Miller: Aplasia Pulmonum, Trudi Obsh. dietsk. vrach., Moscow **3**:32, 1894-95; cited by Hurwitz, S., and Stephens, H. B.: Agenesis of Lung: Review of Literature and Report of Case, Am. J. M. Sc. **193**:81, 1937.

Naclerio, E. A., and Hechberg, L. A.: Congenital Absence of Lung, New York J. Med. **50**:1267, 1950.

Paul, F.: Fehlbildungen im Bereiche der Atmungsorgane, Arch. path. Anat. **267**:295, 1928.

Ponfick: Ein Fall von angeborene primärer Atrophie der rechten Lunge, Arch. path. Anat. **50**:633, 1870.

Purriel, P., and Piaggio, A. A.: Agenesia pulmonar, Arch. urug. med. **14**:9, 1939.

Ratjen, E.: Mittheilungen eines angeborenen Lungenfehlers, Arch. path. Anat. **38**:172, 1867.

Saxl, O.: Beitrag zur Diagnose der Lungenagenesie, Ann. pædiat. **154**:180, 1940.

Sieveking, H.: Über Angeborene und in früher Kindheit erworbene Defektbildungen der Lungen, München. med. Wchnschr. **42**:68, 1895.

Tyson, J.: Congenitally Deficient Left Lung, Philadelphia M. Times **3**:221, 1872-3; Markedly Contracted Kidneys: Uremia; Congenitally Deficient Left Lung, Tr. Path. Soc. Philadelphia **4**:161, 1874.

von Graff, E.: Angeborene Hyperplasie der einen Lunge bei gleichzeitiger Bildung der anderen, München. med. Wchnschr. **52**:598, 1905.

Yampolsky, J., and Fowler, C. D.: Agenesis of Lung: Report of a Case, Arch. Pediat. **55**:293, 1938.

Zipkin, R.: Über ein Adenorrhabdomyom an Stelle der linken und Hypoplasie der rechten Lunge bei einer totgeborenen Frucht, Arch. path. Anat. **187**:244, 1907.

CONGENITAL ABSENCE OF BOTH LUNGS

Allen, and Affelbach: Congenital Absence of Both Lungs, Surg., Gynec. & Obst. **41**:375, 1925.

Roderer, G. J.: Fetus parasitici descriptio, Commentarii Soc. regiae scient. Gottingensis **4**:113, 1754; cited by Schuchhardt, K.: Hochgradige Atrophie (inveterirte Atelectase) der linken Lunge mit compensatorischer Hypertrophie der rechten, Arch. path. Anat. **101**:71, 1885; cited by Schmit, H.: Ein Fall von vollständiger Agenesie beider Lungen, ibid. **134**:25, 1893; cited by Schneider, P., in Schwalbe, E.: Die Morphologie der Missbildungen des Menschen und der Tiere, Jena, G. Fischer, 1909-1913, Vol. 3, Pt. 2, p. 772; cited by Lubarsch, O., and Ostertag, R., editors: Ergebnisse der allgemeinen Pathologie und pathologischen Anatomie des Menschen und Tiere, Wiesbaden, 1896-1923, Vol. 8, Pt. 1, p. 267.

Saito, J.: Ein Foetus ohne Trachea und Lunge, Verhandl. des I. Aerztkongresses in Tokio **4**:2, 1902; cited in Schwalbe, E.: Jahresb. der Anat. u. Entwcklng. **2**:232, 1903; cited by P. Schneider in Schwalbe, E.: Die Morphologie der Missbildungen des Menschen und der Tiere, Jena, G. Fischer, 1909-1913, Vol. 3, Pt. 2, p. 772.

Schmit, H.: Ein Fall von vollständiger Agenesie beider Lungen, Arch. path. Anat. **134**:25, 1893; cited by P. Schneider in Schwalbe, E.: Die Morphologie der Missbildungen des Menschen und der Tiere, Jena, G. Fischer, 1909-1913, Vol. 3, Pt. 2, p. 772.

Coats, J.: A Manual of Pathology, London, Longmans [and others], 1883, p. 513; cited by Neisser, E.: Über einseitige Lungenatrophie und über angeborenen Bronchiektasie, Ztschr. klin. Med. **42**:88, 1901; cited by Schuchhardt, K.: Hochgradige Atrophie (inveterirte Atelectase) der linken Lunge mit compensatorischer Hypertrophie der rechten, Arch. path. Anat. **101**:71, 1885.

de Pozzi, A.: Miscellanea Curiosa medico-physica. dec. I. an. 14, obs. 30, p. 30 (1673-1674) Frankfort-Leipzig; cited by Meckel, J. F.: Handbuch der pathologischen Anatomie, Leipzig, C. H. Reclam, 1812-1816, Vol. 1; cited by Neisser, E.: Über einseitige Lungenatrophie und über angeborene Bronchiektasie, Ztschr. klin. Med. **42**:88, 1900.

Finkelstein, H.: Lehrbuch der Säuglingskrankheiten, Berlin, H. Komfeld, Vol. 2, Pt. 1, 1908, p. 89; Ed. 3, Berlin, Springer-Verlag, 1924, Vol. 3, p. 633; cited by Schneider, P., in Schwalbe, E.: Die Morphologie der Missbildungen des Menschen und der Tiere, Jena, G. Fischer, 1909-1913, Vol. 3, Pt. 2, p. 772.

Klebs, E.: Missbildunge nder Lunge, Ärztl. Korrespondenzbl. Böhmen **2**:111, 1874; cited by Meyer, O.: Über einseitige congenitale Lungenatrophie, Arch. Entwcklngs.-mechn. Organ. **30**:342, 1910.

Meckel, J. F.: Museum der Heilkunde, Zurich, 1794, Vol. 2, p. 204; cited in Handbuch der pathologischen Anatomie, Leipzig, C. H. Reclam, 1812, Vol. 1, p. 477; cited by Fürst, L., in Handbuch der Kinderkrankheiten, edited by C. Gerhardt, Vol. 3, Pt. 2, Tübingen, H. Laupp, 1878, p. 553.

Meyer, O.: Über einseitige congenitale Lungenatrophie, Arch. Entwcklngs.-mechn. Organ. **30**: 342, 1910.

Morgagni, J. B.: The Seats and Causes of Diseases Investigated by Anatomy, translated from the Latin by B. Alexander, London, A. Millas and T. Cadell, 1769, Vol. 4, p. 136.

Neisser, E.: Über einseitige Lungenatrophie und über angeborene Bronchiektasie, Ztschr. klin. Med. **42**:88, 1900.

Otserroht: Verlagerung des Herzens infolge angeborenen Lungendefektes, Deutsche mil.-ärtzl. Ztschr. **39**:431, 1910.

Pruys, W. M.; Veen, R., and Zuidema, P. J.: Case of Pulmonary Aplasia, Geneesk. tijdschr. Nederl.-Indië **79**:398, 1939.

Recklinghausen, cited by Neisser, E.: Über einseitige Lungenatrophie und über angeborene Bronchiektasie, Ztschr. klin. Med. **42**:88, 1900; cited by Schuchhardt, K.: Hochgradige Atrophie (inveterirte Atelectase) der linken Lunge mit compensatorischer Hypertrophie der rechten, Arch. path. Anat. **101**:71, 1885.

Reinhold, H.: Über angeborene und in früher Kindheit erworbene Defektbildungen der Lungen, München, med. Wchnschr. **40**:845, and 869, 1893; cited by Altmann, F.: Zur Kenntnis der Lungenhypoplasien, Ztschr. ges. Anat. **88**:500, 1928.

Riviere: Sepulcr. Anatom., Lib. III, Sec. XVIII, obs. 27 (2); cited by Fürst, L.: Handbuch der Kinderkrankheiten, edited by C. Gerhardt, Vol. 3, Pt. 2, Tübingen, H. Laupp, 1878, p. 553; cited by Schneider, P., in Schwalbe, E.: Morphologie der Missbildungen des Menschen und der Tiere, Jena, G. Fischer, 1909-1913, Vol. 3, Pt. 2, p. 772.

Schlicht: Ein Fall von Missbildungen Beider Lungen, Ärztl. Int.-Bl. München **32**:401, 1885; cited by Neisser, E.: Über einseitige Lungenatrophie und über angeborene Bronchiektasie, Ztschr. klin. Med. **42**:88, 1900.

Schuchhardt, K.: Hochgradige Atrophie (inveterirte Atelectase) der linken Lunge mit compensatorischer Hypertrophie der rechten, Arch. path. Anat. **101**:71, 1885.

Sömmering, J. T.: Supplement, in Baillie: Anat. d. Krankh. Baues, etc., Berlin (1820), p. 445; cited by Meckel, J. F.: Handbuch der pathologischen Anatomie, Leipzig, C. H. Reclam, 1812-1816, Vol. 1; cited by Bohnholtzer, E.: Beitrag zur Kenntnis einseitiger Lungenagenesien, Beitr. path. Anat. **101**:614, 1938.

Thorel: Infantil entwickelte Lunge mit ungewöhnlich starker Hypoplasie des rechten Oberlappens, München. med. Wchnschr. **50**:186, 1903.

Toro, E., and Sa, A.: Malformaciones congénitas del pulmón, Rev. san. y asist. social **9**:1389, 1944.

Wollmann, H.: Ein Fall von Agenesie der linken Lunge mit Bronchiektasien, Dresden, Lehmann, 1891; cited by Neisser, E.: Über einseitige Lungenatrophie und über angeborene Bronchiektasie, Ztschr. klin. Med. **42**:88, 1900.

UNOBTAINABLE REFERENCES

Lukin, M. A.: Two Cases of Entire Absence of Lungs, Med. pribav. k morsk. sborniku, St. Petersburg, p. 423, 1885.

Marino, U.: Agenesia pulmonar unilateral, Rev. tisiol. práct. **2**:120, 1936.

Mazzei, E. S.: Pulmonary and Alveolar Agenesis, Institutode Tisiologia, Universidade nacional de Cordoba, 1942, p. 233.

Snapper, I.: Demonstratie von klinischen Patienten, Verslagen van de Geneeskundige Kring, Amsterdam, Oct. 27, 1936, p. 1.

OTHER REFERENCES

Francke, W.: Über Lungenschrumpfung aus der ersten Lebenszeit: Ein Beitrag zur Kenntniss der atelektatischen Bronchiektasien, Arch. klin. Med. **52**:125, 1893.

Fürst, L.: Missbildungen der Lunge, in Handbuch der Kinderkrankheiten, edited by C. Gerhardt, Vol. 3, Pt. 2, Tübingen, H. Laupp, 1878, p. 553.

Gruenfeld, G. E., and Gray, S. H.: Malformations of the Lung, Arch. Path. **31**:392, 1941.

Hart, C., and Mayer, E., in Henke, F., and Lubarsch, O.: Handbuch der speziellen pathologischen Anatomie und Histologie, Vol. 1, Berlin, Springer-Verlag, 1928, p. 468.

Lawrence, A.: Lung with Abnormal Lobe, in Proceedings of the Anatomical Society of Great Britain and Ireland, J. Anat. & Physiol. **31**:30-31, 1897.

Müller, H., in F. Henke, and Lubarsch, O.: Handbuch der speziellen pathologischen Anatomie und Histologie, Vol. 2, Berlin, Springer-Verlag, 1928, pp. 539, 593.

Schneider, P., in Schwalbe, E.: Die Morphologie der Missbildungen des Menschen und der Tiere, Jena, G. Fischer, 1909-1913, Vol. 3, Pt. 2, p. 772.

van Joost, C. R. N. F.: Three Cases of Congenital Anomalies of Lungs in Children, Geneesk. tijdschr. Nederl.-Indië **79**:1155, 1939.

Obituaries

JOSEPH STILES WALL, M.D.
1876-1952

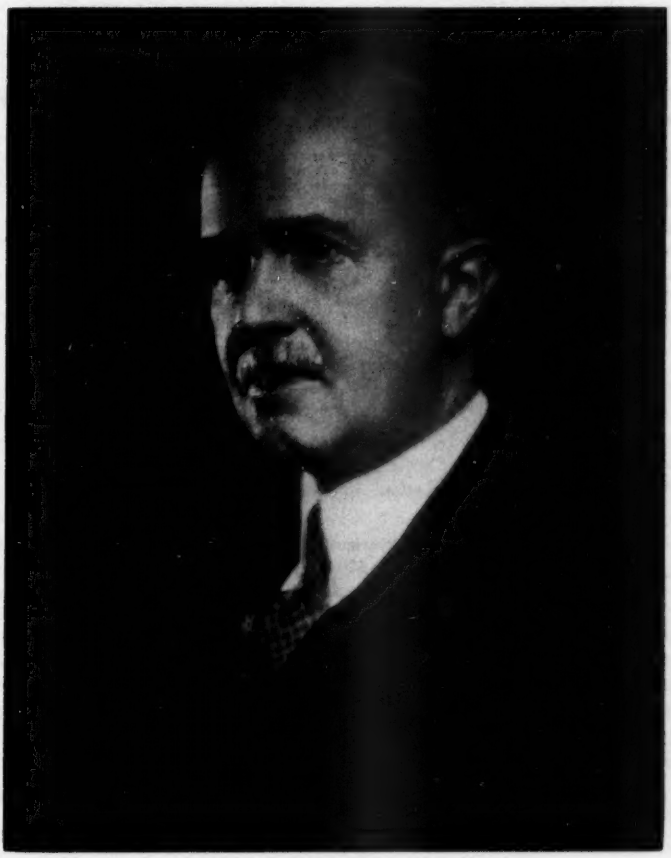
In the death of Joseph Stiles Wall, on Sept. 18, 1952, the pediatric world lost one of its most distinguished members.

Dr. Wall was born in Washington, D. C., Oct. 3, 1876, the son of the late Allen Wall, of Pittsburgh, and Elizabeth Stiles Wall, of Boston. He received his early education in the public schools of Washington, graduating from the Central High School in the class of 1893. He received from that institution three awards, the first, a medal in competitive drill as the best drilled officer of the regiment of cadets in the manual of arms, the second, a scholarship, after competitive examination, to the Georgetown University School of Medicine, which determined his career, and the last, in 1944, a "Certificate of distinction in medicine and social service," by the alumni association.

Georgetown University conferred upon him the degree of Doctor of Medicine, in 1897, the honorary degree of Master of Arts, in 1925, and the honorary degree of Doctor of Science, in 1938. Following postgraduate work in New York and Oxford, England, he engaged in the general practice of medicine for a number of years, later confining his practice exclusively to the field of pediatrics. In subsequent years he became attending pediatrician to Foundlings, Providence, Georgetown University, and Gallinger Municipal Hospitals and consulting pediatrician to Doctors and Columbia hospitals. He was attached to the Children's Hospital for a period of over 50 years, in the last 17 of which he served as chief of staff, retiring in 1951. For 47 years following his graduation in medicine he had taught in the medical school of his alma mater, first as assistant to the professor of chemistry, then as professor of physiology, and finally, for the greater period, as professor of pediatrics.

The Medical Society of the District of Columbia, of which he was president in 1926 and a life member since 1939, twice honored him with scrolls for distinguished services to the profession, the society, and the community. In 1945 he became president of the American Academy of Pediatrics after years of conspicuous accomplishment as chairman of the committee on legislation, for which he was presented a scroll in 1946. Dr. Wall was the recipient of many outstanding awards and citations, in addition to those mentioned, among which was a letter from the Honorable Herbert Hoover, then president of the American Child Health Association, welcoming and commending him as a newly appointed director of that organization in 1923. In 1944 he was elected president of the American Academy of Pediatrics. In 1947, in recognition of his efforts in cementing friendly and intimate relationships among the pediatricians of North America and those of the Latin-American Republics, President Grau Martin of Cuba conferred upon him the Decoration of Merit, Order of Carlos L. Finlay, grade of commander. Dr. Wall was affiliated with the

American Medical Association, the Southern Medical Association, the American Public Health Association, and the Washington Academy of Sciences. He was an honorary member of the Sociedad de Pediatria de Cuba and the Latin-American division of the Academy of Pediatrics. He served for many years on the Advisory Committee on Maternal and Child Health to the U. S. Children's Bureau.



JOSEPH STILES WALL, M.D.
1876-1952

Dr. Wall was truly a great physician who leaves behind an enduring memory of a life devoted to service.

Surviving Dr. Wall is a daughter, Mrs. Frances Wall Hines. The death of his beloved wife, Agnes, preceded his own by just 45 days, the duration of his final illness.

Abstracts from Current Literature

Metabolism; Infant Feeding; Milk and Other Foods

COBALT METABOLISM OF YOUNG COLLEGE WOMEN ON SELF-SELECTED DIETS. MARY JONES HARP and FLORENCE I. SCOULAR, J. Nutrition **47**:67 (May 10) 1952.

The average daily intake and excretion of cobalt of 23 young college women was studied by Mary Jones Harp and Florence I. Scoular. Twenty-one of the 23 subjects were in positive cobalt balance on these self-selected diets. From 73 to 97% of the dietary cobalt was absorbed. The main pathway of excretion was through the urine. There were just two negative balances, and in each case these balances were negative because of higher urinary cobalt; nor were the two negative balances due to an inability to absorb cobalt from the intestinal tract, since these subjects had absorption percentages of 92 and 94, respectively, as compared with the range of 73 to 97% for the entire group of subjects.

FREDEEN, Kansas City, Mo.

ADEQUACY OF ARTIFICIAL FEEDING IN INFANTS. S. S. STEVENSON, J. Pediat. **31**:616 (Dec.) 1947.

Having prefaced his remarks by the statement that the natural and preferred way to feed the human infant is at his mother's breast, if this is feasible and practical, the author continues by briefly discussing the major objections to cow's milk as food. He holds none of these to be valid.

In presenting the records of 263 infants studied by him, the author makes reference to the work of Grulee and Ebbs in the advocacy of breast feeding. On the basis of his studies, the author concludes that there is an increased amount of respiratory infection in the second half year of life in the artificially fed infant as compared with the breast-fed. This difference is significant but not great. It is suggested that the difference is probably explainable on the basis of greater vitamin A and vitamin C intake combined with better utilization or decreased need in the breast-fed infant.

Two hundred twenty international units of vitamin A per pound of body weight (440 I.U. per kilogram) daily and 100 mg. of ascorbic acid daily (4 oz. [113 gm.] orange juice) in several divided doses may correct the greater tendency for respiratory infection in the artificially fed infant.

GRULEE, Galveston, Texas.

EFFECT OF CORTISONE ON NUTRITION. LEO J. GEPPERT and BERNARD F. O'HARA, J. Pediat. **39**:267 (Sept.) 1951.

When cortisone is given for a long time it is antianabolic. Given to older children for ten days or less it causes increased appetite and nitrogen retention. Geppert and O'Hara suggest short intermittent periods of cortisone therapy for nutritional failure. Results in newborn infants were erratic.

MACDONALD, Pittsburgh.

USE OF ASCORBIC ACID TABLETS TO ENRICH MILK FOR INFANT FEEDING. ARTHUR D. HOLMES and CARLETON P. JONES, J. Pediat. **39**:320 (Sept.) 1951.

The tablet used contains crystalline ascorbic acid, a filler (lactose), a disintegrator (cornstarch), a binder (acacia, gelatin, or glucose), and a lubricant to prevent sticking (purified talc, white petrolatum, calcium stearate, or dried starch). It dissolves quickly when dropped into the infant's formula.

MACDONALD, Pittsburgh.

STUDY ON ORANGE JUICE, ORANGE JUICE CONCENTRATE AND ORANGE PEEL OIL IN INFANTS AND CHILDREN. C. L. JOSLIN and J. E. BRADLEY, J. Pediat. **39**:325 (Sept.) 1951.

Orange juice, fresh or frozen, is a good source of vitamin C. It seldom causes skin eruptions or gastrointestinal disturbances in infants. If symptoms of this sort occur, they may be caused by peel oils which are more likely to be irritating. The best way to extract the juice is with an electric juicer, rather than with a pressure squeezer, which is likely to squeeze out peel oils. One should start at age 3 weeks with ¼ oz. (7 gm.) diluted orange juice, and increase gradually.

MACDONALD, Pittsburgh.

SUBCUTANEOUS ADMINISTRATION OF COMBINED FAT EMULSION WITH HYALURONIDASE. B. G. P. SHAFIROFF, H. C. BARON, J. RECHT, and JOHN H. MULHOLLAND, Proc. Soc. Exper. Biol. & Med. **77**:608 (Aug.) 1951.

Hyaluronidase was added to a 10 per cent combined fat emulsion containing 100 gm. of purified coconut oil, 50 gm. of a protein hydrolysate, 50 gm. of dextrose, and 30 gm. of gelatin, which provided approximately 1,200 Cal. per liter. The particle size of the emulsion averaged 1 μ. Hyaluronidase in doses of 500 units was injected into the thigh of 20 patients; then the emulsion was infused into the subdermal tissue at the rate of from 4 to 7 ml. per minute. The subcutaneous infusion of one liter of the 10% fat emulsion was well tolerated by all. In 86% of the patients there was a temperature elevation of 1 F. to 2 F. and in some instances more, but in all subjects the temperature returned to its usual pattern at the end of the study period. There were no significant changes in the blood pressure. The total blood lipids reached a peak level in one-half to four hours after the clysis was begun, and the peak level averaged twice that of the fasting level. About 10 to 15 mg. of lipid was recovered from the urine except in one instance; in a patient with severe kidney disease, excretion was 69 mg. Oxygen consumption rose slowly, the average maximum increase in oxygen uptake being 20%. There was no evidence of a toxic systemic reaction or of any pathologic change at the site of the injection. The authors feel that their study indicates that a combined fat emulsion together with hyaluronidase can be administered successfully for nutritive purposes via the subcutaneous route.

HANSEN, Galveston, Texas.

THE MATERNAL BREAST. DAVID FINDLEY, South. M. J. **45**:51 (Jan.) 1952.

Nonorganic factors which seem to discourage mothers from nursing their infants at the present time include socioeconomic factors, community activities, vanity, psychogenic factors, exhaustion, insufficient encouragement by the physician, and ignorance on the part of the mother about the value of breast feeding. Suggested remedies for this are education, adequate prenatal care, proper postpartum care (though the reviewer cannot agree with the necessity of the recommended "toughening up" and cleansing of nipple procedures), and a good diet.

LOGAN, Rochester, Minn.

TREATMENT OF DEHYDRATION IN INFANCY USING CONTINUOUS INTERSTITIALS AND HYALURONIDASE. ALICE GOODFELLOW, RUTH POCOCK, and JOHN MCCREARY, Canad. M. A. J. **66**:8 (Jan.) 1952.

By using hyaluronidase, hydration was reestablished in most cases as quickly by using interstitial or subcutaneous drip as by using intravenous administration. Electrolyte balance was easily achieved. Severely dehydrated patients with peripheral vascular collapse require intravenous fluids for the first twenty-four hours.

MCGUIGAN, Evanston, Ill.

MALNUTRITION IN EGYPTIAN INFANTS. MAMDOUH HANAFY, J. Royal Egyptian M. A. **34**:470, 1951.

The general picture of undernutrition in Egyptian infants under 2 years is similar to the syndrome of Kwashiorkor. All patients have a pellagrous rash, acrodynia, cirrhosis of the liver, or a syndrome resembling von Jaksch's, with wasting. The deficiencies are varied, mainly

protein, vitamin, and mineral deficiencies, with eventual endocrine dysfunction. Variations in the picture depend on degree and relative variation in the deficiencies mentioned. A high-protein diet reinforced with vitamins and minerals is usually effective. McGuigan, Evanston, Ill.

Egg Soup as a Nourishment for Infants. K. Geiger, Ann. pædiat. **178**:83 (Feb.) 1952.

A description is given of a baby food prepared from egg yolk, flour decoction, and sugar, with the addition of a small quantity of sodium chloride and calcium gluconate. Physiological investigations and clinical observations in infants who were fed with this food are reported. The chief indication is the feeding of older infants who are dystrophic and show lack of appetite. The food is contraindicated in allergic and dyspeptic conditions. The food does not contain milk, and yields 102 Cal. per 100 cc. From the Author's Summary.

Chestnut Diet. A. Solé, Ann. pædiat. **178**:98 (Feb.) 1952.

An exclusive chestnut diet (the composition of which is given in detail) has shown good results in the treatment of celiac disease and gastroenteritis in infants and older children, and also in cases of infantile eczema. From the Author's Summary.

Beta Lactose in Infant Feeding. G. Calzetti, Clin. pediat. **29**:659 (Nov.) 1947.

Very satisfactory clinical results followed the use of beta lactose as the added carbohydrate in the formulas of healthy, of dystrophic, and of premature infants. Higgins, Boston.

Amino Acids and Absorption of Iron. L. Salmi, Clin. pediat. **30**:587 (Sept.) 1948.

Two tests were made with each of 10 children. In the first test 1 gm. of reduced iron was given by mouth to the fasting child; blood-iron determinations were made before and three, six, and eight hours after the iron was eaten. In the second test, the same procedure was followed, except that 5 gm. of amino acids (hydrolyzed casein) was given at the same time as the reduced iron. In the first test, the blood iron increased from 101 γ to 142 γ per 100 cc. in three hours and had fallen to the control values by the end of six hours. In the second test the blood iron increased from 102 γ to 227 γ per 100 cc. in three hours and was still elevated (143 γ) after eight hours.

Higgins, Boston.

Nicotinamide and Hypogalactia. L. Martoni, Clin. pediat. **33**:496 (Aug.) 1951.

Forty nursing mothers with poor breast-milk supply were given 0.3 gm. nicotinic acid daily for periods of 15 to 20 days. About 50% of the mothers had a very gratifying increase in breast-milk production. The suggestion is made that the vitamin caused an increased blood supply to the mammary tissue and to the endocrine glands, and hence increased lactation.

Higgins, Boston.

Relation of the Weather to the Frequency of Toxicosis in Infants. L. Martoni, Clin. pediat. **33**:687 (Nov.) 1951.

The average temperature, barometric pressure, and humidity were plotted for each day in the months when there was a high incidence of toxic diarrhea in infants in Bologna. No definite connection between the meteorologic conditions and the incidence of the disease could be found.

Higgins, Boston.

Beta Lactose in the Artificial Feeding of the Dystrophic Infant. I. Traina and C. Severi, Lattante **18**:657 (Nov.) 1947.

Observations were made on 17 hospitalized infants who presented nutritional problems. The efficacy of beta lactose as the added carbohydrate of a cow's milk formula was tested.

The conclusion was drawn that beta lactose contributed to a change of the intestinal physiology so as to result in stools which approximated those of healthy children. This sugar appears particularly indicated in constipation and in protein indigestion.

HIGGINS, Boston.

STREPTOMYCIN THERAPY IN ACUTE AND CHRONIC NUTRITIONAL DISTURBANCES IN HOSPITAL PRACTICE. E. ZECCA, Lattante **22**:536 (Sept.) 1951.

Twenty-two infants with acute enteritis and three with chronic malnutrition were treated with streptomycin parenterally (0.04 gm. per kilogram per day). One infant died, 10 showed improvement and 14 were completely cured. The infants began immediately to show marked weight gains.

HIGGINS, Boston.

TECHNIQUE IN RESUMING FEEDING OF TOXIC INFANTS. M. NEGRI, Lattante **22**:596 (Oct.) 1951.

Negri recommends that the feedings of infants with toxic diarrhea be started only after the initial treatments with antibiotics and blood plasma. He recommends that no milk be given for several days, and feeds mainly carbohydrate in the form of rice. At first a 2% rice water is offered; the rice content is gradually increased, first to the consistency of a gruel and later to a thick porridge.

HIGGINS, Boston.

DISCUSSION REGARDING THE BASAL METABOLISM OF CHILDREN. C. CISI, Minerva pediat. **3**:509 (Sept.) 1951.

The low basal metabolism of newborn infants may be ascribed to slow establishment of body-temperature regulation. The basal metabolism falls rapidly as the child approaches the age of 18 months. In estimating the caloric requirement of the child, it is necessary to take into consideration the calories necessary for the formation of new body tissue.

HIGGINS, Boston.

CONCENTRATED HUMAN MILK. ALESSANDRO BIEBER, Riv. di clin. pediat. **45**:577 (Oct.) 1947.

Human milk is the ideal food for the normal infant, both in the physiological and the pathological field, but is not always sufficient to solve the feeding problem of the premature infant, the newborn underweight infant and the atrophic infant who need a relatively greater caloric intake and have different and complex biological needs. The author prepares human milk in such a way as to include in a reduced volume the necessary requisites for a harmonious growth. With a technique originated by the author which consists in evaporating human milk in a vacuum at a fixed temperature of 55 C., half-concentrated human milk is obtained, the biochemical elements of which have in no way been altered. With this type of nourishment, perfectly tolerated in every case, infant feeding (maternal, mixed, or artificial) is integrated in cases of premature, congenitally weak, underweight newborn, and atrophic infants for whom the feeding problem could not be solved by any other means. Of the patients treated in more than two years of study, of whom 22 are reported on, there were 18 premature infants, 11 born two months before term, 7 one month before term, 3 atrophic infants born at term, and also a weak infant with congenital anencephaly; these infants were of various ages and weighed about 1,500 gm. and sometimes less. It has been observed that when even very small quantities of half concentrated human milk (from 30 to 100 gm. per day divided in meals) is given a satisfactory increase in the weight curve is always noted, so that a loss of weight or a stationary weight curve due to various and complex causes ceases. The use of half-concentrated human milk is proposed for all those cases in which there are difficulties due to the calorimetric and qualitative needs of the infant, so as to overcome the vicious circle of the hunger ration and rapidly arrive at the growth ration and then at the best feeding formula. The biological significance of the concentrated human milk employed by the author is pointed out, especially for the high concentration of the amino acids essential to growth and of fundamental importance in the complex metabolic processes of an organism during growth, for their direct action and through stimulation of the glands of internal secretion.

FROM THE AUTHOR'S SUMMARY.

CORTISONE IN ACUTE NUTRITIONAL DISORDERS OF INFANCY. FEDERICO C. MILIA and JOSE LAMELAS, Arch. argent. pediat. **36**:82 (Aug.) 1951.

Seven infants with severe diarrhea were given small amounts of cortisone in addition to the full regimen ordinarily employed. One died; the others made remarkable recoveries. A footnote adds 38 cases in which similar treatment was given, with 7 deaths.

PANOS, Galveston, Texas.

STUDIES ON THE DYSTROPHIC INFANT: II. POSTMORTEM DETERMINATIONS OF THE LIPIDS of the Liver. HERMANN NIEMEYER and SEGISMUNDO ITURRA T., Rev. chilena de pediat. **18**:141 (March) 1947.

Postmortem lipid determinations were made in 50 cases of undernourished infants. Twenty-seven infants had suffered dehydration (toxicosis) with or without secondary infections and 23 died as a result of various chronic and acute diseases.

A description of total lipid and phospholipid correlations is given. When total lipids increased, an increment of phospholipids that is not directly proportional was observed. At first a small increment of total lipids was followed by big variations in phospholipids, but phospholipids had a tendency to reach a maximum level while total lipids continued increasing.

Cholesterol follows a directly proportional increase with total lipids.

Lipid and phospholipid rates were very frequently grouped close to the correlation curve described, when no especial group in any one of the acute diseases was observed. In chronic diseases or in cachexia they tended to group in the first part of the curve.

A correlation was found between the results of chemical and of anatomicopathological examinations in that with extensive fatty degeneration an increase of lipids rate was found.

The percentage that phospholipids represent in total lipids had a maximum level in mild fatty degeneration and tended to decrease as soon as the degeneration increased. This phenomenon is explained as a compensatory mechanism that tends to mobilize the liver fats.

AUTHORS' SUMMARY.

ANIMAL EXPERIMENTATION TO DETERMINE THE PATHOGENESIS OF PARENTERALLY CAUSED TOXICOSIS OF INFANTS. K. STENGER, Ztschr. Kinderh. **67**:639, 1949-1950.

The author produced by chemical irritation of the middle ear toxicosis-like symptoms in animals and contends that the toxicosis in infants is due not to bacteria or bacterial products but rather to neurogenic gastrointestinal disturbances caused by inflammatory irritation of the mucosa of the middle ear.

GLASER, Jerusalem.

SIMULTANEOUS INVESTIGATION OF THE MANIFESTATIONS OF SUBMINIMAL NUTRITION ON URBAN AND RURAL CHILDREN. W. AUERSWALD and H. BORNSCHEIN, Wien. klin. Wchnschr. **52**:854 (Dec. 31) 1947.

The authors report a comparative study on the effects of a low-caloric diet on urban and rural children. Simultaneous investigations of 300 school children, 150 urban and 150 rural, on low-caloric diets yielded the following results:

The majority of the children investigated in both groups were underweight. In the urban group there was a drop of serum protein, of erythrocytes, and of hemoglobin to low normal levels, while the rural children showed normal values. The findings indicate a quantitative caloric undernutrition for both groups and a high-grade qualitative undernutrition for the urban children. The mental fatigue of the urban group, as compared with the rural children, was obviously increased. The changes in biologic data and decreased ability to concentrate indicate a common source of deficiency as the causative factor, namely, a marked deficiency in protein.

ABT, Chicago.

ROLE OF WATER DEFICIENCY IN THE PATHOGENESIS OF TOXIC METABOLIC DISORDERS IN INFANCY. Ö. KERPED-FRONIUS, Orvosok lapja **3**:1661 (Oct. 26) 1947.

Exsiccation due to thirst should be differentiated from exsiccosis following primary salt deficiency. The first form is associated with the increase of salt concentration; the water loss

is divided between the extracellular and the intracellular fluid system. For this reason anhydremia appears only in the case of considerable absolute water loss. In the case of exsiccosis attending salt deficiency, anhydremia appears after a water loss representing 10% of the body weight. "Toxicosis" is a secondary state, being essentially the pattern of infantile shock, attending water loss and enteral and parenteral infections. The circulatory disorder may be caused by the decrease of the quantity of blood plasma, due to salt and water loss. In a great number, however, the circulatory disorder is only partly of anhydremic origin. The products of metabolism of the infecting germs directly damage the capillaries, i. e., the circulation, thereby rendering the prospects of the simple salt and water economy more unfavorable. The circulatory disorder exercises a secondary, deteriorating effect on the brain, as well as on the metabolism of the cells; in this injury to the cells the potassium loss also has its role.

GÖRGÉNYI-GÖTTCHE, Budapest, Hungary.

OUR EXPERIENCE IN THE REHYDRATION AND PARENTERAL FEEDING OF INFANTS DURING THE LAST FOUR YEARS. M. JADRNICEK and B. OSTADALOVA, Pediat. listy **6**:341, 1951.

During the four years from 1947 to 1950, in which approximately equal numbers of infants were treated for diarrhea, the proportion of severe cases (severe diarrhea and intoxication) increased from one-third to two-thirds. The high death rate may be explained partly by the high proportion of infants which arrived at the hospital moribund and partly by the large number of infants from outside districts. The death rate fell by an average of 14.5% in 1950 as compared with 1949 as a result of parenteral feeding. During the four years the number of cases of diarrhea arising secondarily to other conditions increased, while the number of complications fell. The number of deaths from marasmus and toxic inanition fell from two-thirds to one-third, but the number due to sepsis rose. If parenteral alimentation does not prove sufficient to renew the weakened resistance of the intoxicated organism it is necessary to use antibiotics.

FROM THE AUTHORS' SUMMARY.

Vitamins; Avitaminoses

HYPERVITAMINOSIS A: REPORT OF AN UNUSUAL CASE WITH MARKED CRANIOTABES. JAY M. ARENA, PAUL SARAZEN JR., and GEORGE J. BAYLIN, Pediatrics **8**:788 (Dec.) 1951.

An unusual case of hypervitaminosis A with severe craniotabes in a 6½-month-old infant is presented. This is the youngest infant reported with this condition, and the first to present craniotabes. Symptoms of hypervitaminosis A are compared with those of hypervitaminosis D and with those of infantile cortical hyperostosis. In the case presented prompt and complete disappearance of craniotabes and other symptoms followed withdrawal of administration of vitamin A.

FROM THE AUTHORS' SUMMARY.

HYPERVITAMINOSIS D IN INFANCY. A. BALLABRIGA and DR. ZAMORA, Arch. Pediat. **1**:175 (Sept.) 1950.

The authors review the literature on the cases of hypervitaminosis due to excessive doses of vitamin D. There seems to be a wide individual difference in tolerance for overdosage with vitamin D. The dose considered by Reed and his collaborators is 20,000 I. U. per kilogram per day. Other investigators have found signs of illness when dosages vary from 25,000 I. U. daily to 150,000 I. U. daily, regardless of weight. Many of the fatal cases reported synthetic concentrates had been taken.

Clinically, anorexia, vomiting, and constipation will develop, then rapid loss of weight, with pallor and even cachexia, and, lastly, renal and nervous system manifestations. Urinary symptoms are polyuria, polydypsia, excessive tricalcium phosphate crystals, albuminuria, microscopic evidence of blood, and cylindruria. Arterial hypertension may be present even in young infants.

Kidney function may be impaired. The blood chemistry may show calcium as high as 14 to 24 mg. per 100 cc. of blood. There may also be hyperphosphoremia.

The pallor may be associated with a hypochromic anemia similar to that associated with toxicity due to heavy metals such as lead. The action on the glomerulus also appears similar to the toxic effects of heavy metals.

The nervous system symptoms may vary from apathy to convulsions, as well as mental confusion and hyperesthesias.

There may be calcium deposits in the arteries and other organs.

Cardiac symptoms may be found such as tachycardia; shortening of the Q T interval and flattening of the T_1 and T_2 waves in the electrocardiograms.

The treatment should include administration of vitamins A and B and enough ammonium chloride to keep the urine alkaline. Dehydration should also be treated. Naturally, the vitamin D should be discontinued for a while.

ALLEN, San Francisco.

HISTOPHYSIOLOGICAL CONTRIBUTIONS TO THE STUDY OF VITAMIN C_2. M. GABE and J.-L. PARROT, Presse méd. **59**:1740 (Dec. 25) 1951.

The use of ascorbic acid alone did not prevent the appearance of scorbutic changes in guinea pigs. The necessity of adding vitamin C_2 was postulated, and photomicrographs are presented supporting this view.

DAFFINEE, Melrose, Mass.

Hygiene; Growth and Nutrition; Public Health

TABLES FOR PREDICTING ADULT HEIGHT FROM SKELETAL AGE. NANCY BAYLEY and SAMUEL R. PINNEAU, J. Pediat. **40**:423 (April) 1952.

In 1946, Nancy Bayley published "Tables for Predicting Adult Height from Present Height and Skeletal Age." This was based on the Todd Atlas. The original Todd Atlas has been revised, and Bayley and Pinneau, in this report, revise the prediction tables.

MACDONALD, Pittsburgh.

INVESTIGATION OF STILLBIRTHS AND DEATHS OF CHILDREN UNDER FIVE YEARS OF AGE. M. VERA BÜHRMANN, South African M. J. **26**:835 (Oct. 18) 1952.

Study of causes of death in Pretoria revealed a disturbing lack of accurate statistics. It was found that environmental health factors cause a smaller proportion of deaths than is commonly supposed. The main causes of death were prematurity, diarrheal diseases, pulmonary infections, and malnutrition. All of these had once been attributed to environmental conditions. This investigation showed that parental ignorance, unwillingness to cooperate, and often apparently wilful neglect also play a very important part.

The study suggests that much of the loss of life through high infant mortality is preventable. Committees such as those on maternal and infant mortality in various South African cities would be of great help here.

MCGUIGAN, Evanston, Ill.

METHODS FOR EVALUATING PHYSICAL GROWTH. G. COTELLESSA and P. SALOMONE, Clin. pediat. **33**:669 (Nov.) 1951.

The method of using the Wetzel grid is explained, as it is not well known in Italy. The relative advantages and defects of the Wetzel grid, the Correnti auxogram and the De Toni auxometric grid are discussed. De Toni's grid is preferred as the most practical.

HIGGINS, Boston.

INFLUENCE OF SULFONAMIDES AND ANTIBIOTICS ON DENOGRAPHY. PEDRO LEON LUQUE, Arch. argent. pediat. **36**:77 (Aug.) 1951.

A sharp decrease in general and infant mortality rates in the province of Córdoba followed the introduction of the multiple sulfonamide preparation in 1939. Antibiotics have furthered this reduction to a much smaller degree. Principal causes of death were also markedly affected, pneumonia having been replaced by cardiopathies as the outstanding cause in 1945, at which time it ranked fourth, and diarrheas fifth. The average age of death rose from 32 in 1936 to 47 in 1948. The influence of improved general hygienic measures is discussed briefly.

PANOS, Galveston, Texas.

Prematurity and Congenital Deformities

MATERNAL SYNDROME ASSOCIATED WITH ISOIMMUNIZATION AND FETAL DEATH IN UTERO. Irwin H. KAISER, Minnesota Med. **35**:25 (Jan.) 1952.

Three case histories are given in which isoimmunization seemed to produce a definite clinical syndrome in the mothers. The clinical picture is characterized by a previous history of Rh incompatibility and the appearance of sudden weight gain and manifest edema with subsequent fetal death in utero. No explanation as to etiology was given.

STOESSER, Minneapolis.

CASE OF PARASITIC SIAMESE TWINNING. N. J. Roos and L. J. DUPREEZ, South African M. J. **26**:46 (Jan. 19) 1952.

A case of hydramnios resulting in Caesarean section is described. The infant was found to have attached to the head an enormous teratoma constituting a parasitic Siamese twin. Operative removal was successful.

McGUIGAN, Evanston, Ill.

FOETUS PAPYRACEUS. R. St. C. SINCLAIR, South African M. J. **26**:69 (Jan. 26) 1952.

The author presents two cases of fetus papyraceus and discusses possible causes of intrauterine death.

McGUIGAN, Evanston, Ill.

HARE-LIP AND CLEFT PALATE. EUSTACE J. DE SOUZA, Indian J. Child Health **1**:443 (Sept.) 1952.

Etiology, incidence and surgical techniques are discussed in a general consideration of harelip and cleft palate. The surgical management is considered at length, attention being given to time of operation, preoperative care, and anesthesia. Ether administered intrathecally is the anesthetic of choice. Postoperatively, local block of the facial nerve, lasting two to three weeks, is an effective aid in providing rest for the suture line.

McGUIGAN, Evanston, Ill.

FAMILIAL DYSCHONDROSTEROSIS (OSTEOCHONDRODYSTROPHIA DEFORMANS). M. KAPLAN, É. GUY and A. CANTAGREL, Presse méd. **59**:1723 (Dec. 25) 1951.

In a girl of 14 years pains developed in the wrists, which the mother had considered deformed from early infancy. Examination showed bony deformities of the forearms and the legs which were typical of the form of osteochondrodystrophy associated with dwarfism.

The mother of this child was affected similarly, and also the maternal grandfather, a maternal great-aunt, and the maternal great-grandfather.

DAFFINEE, Melrose, Mass.

PROGNOSIS OF PREMATURITY IN FIRST YEAR OF LIFE. E. MOSER, Ann. paediat. **178**:1 (Jan.) 1952.

A report is made on 1,368 premature infants admitted to the babies' home of Berne canton. As more premature infants who were underweight or otherwise severely damaged were admitted, the absolute and relative number was increased. The most important factor which led to premature birth was maternal toxemia. The average mortality of premature children was 22.8%. There was increase of mortality in the newborn period and marked decrease of late mortality. More than one-third of the deaths in the newborn period happened on the first day of life. The most important cause for the early mortality was the causal sequence debility-intracranial-hemorrhage-asphyxia, and for the late mortality, interstitial pneumonia of the premature child during the first trimenon. Statistical data of the weight development of the premature infants are given.

FROM THE AUTHOR'S SUMMARY.

NEW IDEAS AS TO GIGANTISM IN CHILDREN. S. STEFANINI, Clin. pediat. **33**:555 (Sept.) 1951.

The record is presented of a boy aged 2 years 3 months who was the size of a 7-year-old child. He weighed 21.7 kg., and was 115.5 cm. tall. His facies was that of a 12-year-old boy, and x-ray examination revealed a bone age of 10 years. His skin was markedly pigmented where

it was exposed to light; there was abundant pubic hair and there were enlarging genitalia. The blood pressure was 195/145. The child had continuously a febrile temperature, which was unaffected by salicylates or penicillin. The eyegrounds were normal.

The family history revealed that two of the six siblings were hermaphrodites. The suggestion is made that the condition might be due to unusual hereditary embryological elements, ultimately involving the pituitary, adrenal, pineal, and testicular glands.

HIGGINS, Boston.

UNUSUAL CASE OF MULTIPLE CONGENITAL MALFORMATIONS. U. GIURANNA, Lattante **22**:547 (Sept.) 1951.

An infant, dying on the fourth day of life, had the following malformations—arhinoencephaly, microphthalmos, atresia of the right auditory canal, and malformed right auricle. The abnormalities are somewhat analogous to some reported congenital malformations in animals traceable to vitamin A and B_9 deficiency.

HIGGINS, Boston.

BONNEVIE-ULLRICH STATUS. C. ZUNIN, Minerva pediat. **3**:547 (Oct.) 1951.

The Bonnevie-Ullrich status covers a group of cases of multiple malformations. It has also been called Turner's syndrome, and includes the Ehlers-Danlos syndrome of multiple congenital arthrogryposis. Five cases from the Genoa Clinic and 3 other Italian cases are analyzed. Pterygia, or webbing, of the neck, axilla, and knee were the common abnormalities in the cases presented. In addition to malformation, there were frequent degenerative stigmata located in the external ear, the eye, the palate, and the skin.

HIGGINS, Boston.

MORQUIO'S DISEASE: CLINICAL AND ROENTGENOGRAPHIC DIAGNOSIS. LORENZO EXPOSITO, ARGELIO DE FERIA, and HUGH GONZALEZ, Rev. cubana de pediat. **19**:447 (Aug.) 1947.

The authors report two patients with abnormal skeletal development, corresponding to those patients with Morquio's disease. The first, a white girl of 13 years, complained of pains in the joints of the lower extremities and an unstable gait. She was short for her age and had thoracic deformities, including kyphosis. Two other members of the family presented similar signs. The second patient, a Negro boy of 9 years 6 months, was small for his age with disproportionately long arms, short trunk, and short neck, and enlargement of the wrists, elbows, and knees. Roentgenograms showed similar changes in both cases.

KEITH, Rochester, Minn.

ACROCEPHALOSYNDACTYLY, OR APERT'S DISEASE. LORENZO EXPÓSITO, Rev. cubana de pediat. **23**:45 (Jan.) 1951.

A typical case of acrocephalosyndactyly in a 13-month-old girl is presented. The authors report this as the second published case from Cuba. Clinical description, photographs, and x-rays are given.

NELSON, San Francisco.

SERUM PROTEINS IN PREMATURE INFANTS. OSCAR CORREA and HUMBERTO DEL POZO, Rev. chilena pediat. **12**:544 (Dec.) 1950.

The authors report studies of total serum proteins on 109 healthy premature infants, whose weight was between 1,600 and 2,500 gm. and whose weight curves were estimated as normal for them. The serum proteins were determined by refractometric procedure.

A mean value of total serum proteins of 56.4 gm. ($\pm$ 6.8) per 1,000 cc. was found. The comparison of this figure with that encountered in a previous work on normal full-term infants (58.9 ± 8.9) gives a difference which is statistically significant. These results are briefly discussed.

FROM THE AUTHORS' SUMMARY.

IMMATURITY AND PREMATURITY. ALMEIDA GOUVEA, Arq. pediat. **22**:29 (June) 1950.

The author gives a comprehensive review of the problem of prematurity and discusses the management of the immature infant.

CHRISTOPHERSON, Wilmette, Ill.

CONGENITAL MICROGNATHIA WITH GLOSSOPTOSIS AND MICROGLOSSIA ASSOCIATED WITH DEXTROCARDIA AND HETEROTAXIA. JULIO C. BELTRAN, Arch. pediat. Uruguay **21**:505 (July) 1950.

The author reports a case of micrognathia in a 2-month-old male infant, in whom the malformation was accompanied by glossoptosis and microglossia and dextrocardia with heterotaxia, a hitherto undescribed association. The patient presented the clinical picture usual in these cases, i. e., crises of dyspnea and cyanosis, high respiratory obstruction, and repeated respiratory infections; in the course of one he was hospitalized. First, the treatment of the respiratory infection was instituted, and then the micrognathia was treated, with posture in ventral decubitus and orthostatic feeding, as recommended by Pierre Robin. Thereafter, Davis and Dunn's apparatus was applied, and after one month of treatment the patient was discharged definitely improved. When the infant was 7 months old, he was found to be in good general condition, and the functional respiratory disorders had disappeared. The author gives a review of the literature, stating that the study of the factors considered responsible by the different authors for the micrognathia does not disclose in the present case what might have been the cause of the malformation. He abstracts all cases published.

FROM THE AUTHOR'S SUMMARY.

NUTRITION OF PREMATURE INFANTS WITH AMINO ACIDS. H. HENCKEL, Ztschr. Kinderh. **67**:221, 1949.

The author added a protein hydrolysate in concentrations of 0.5 to 1% of breast milk and found that it could readily be tolerated by premature infants and that the weight increase of these infants was superior to that of infants fed without the addition of the protein hydrolysate.

GLASER, Jerusalem.

CRANIOSTENOSIS IN CHILDHOOD. K. BRACHFELD and J. SVATY, Pediat. listy **6**:349, 1951.

Five cases of Crouzon's disease are described, two of these in 3-month-old infants. The importance of an early diagnosis is stressed and, if necessary, of operative procedures.

FROM THE AUTHORS' SUMMARY.

RETROLENTAL FIBROPLASIA. EJLER HOLM, Ugesk. laeger **112**:1186 (Aug. 28) 1950.

The author reports 3 cases of retrolental fibroplasia in premature infants of 1,100 to 1,400 gm. birth weight.

WALLGREN, Stockholm, Sweden.

A STUDY ON HEREDITARY TENDENCIES OF HARE LIP AND CLEFT PALATE. YUSHI TOGUCHI and KO NAGAI, Otolaryngology (Japan) **24**:240 (May) 1952.

Toguchi and Nagai investigated 156 cases of harelip and cleft palate. The patients were 83 men and 73 women. There were 77 instances of combined involvement of harelip and cleft palate, 32 cases of lone involvement of harelip, and 47 of cleft palate.

There was definite evidence of consanguinity in these 156 cases. These involved other congenital deformities. There were 10 instances of inguinal hernia, 8 of umbilical hernia, 1 each of microtia and polyotia, and 1 of deaf-mutism.

In 24 instances close relatives of the 156 patients showed harelip or cleft palate or both. In 17 instances marriage between consanguineous persons was traced. Other factors that might have influenced the malformation at the time of conception and during gestation were not established.

HARA, Los Angeles [A. M. A. ARCH. OTOLARYNG.].

Newborn

EXCHANGE TRANSFUSION FOR FETAL ERYTHROBLASTOSIS. HENRY W. KAESSLER and JAMES LEDGARD, J. Pediat. **39**:174 (Aug.) 1951.

Exchange transfusion with compatible nonantigenic blood is the accepted treatment for severe erythroblastosis. The umbilical vein is preferred; if the catheter cannot be introduced into the umbilical vein, or, after 12 to 24 hours, when the umbilical vein may be thrombosed, the great saphenous vein may be used. The authors detail the technique.

The following indications during pregnancy may lead to the need for exchange transfusion: Rh (d) negative mother, 92%; Rh (D) positive father; ABO or other incompatibility; positive indirect Coombs test in first trimester (this may mean severe involvement); increasing Coombs test titer in second and third trimesters; a history of previous sensitizing pregnancy, and a history of previous administration of sensitizing blood. Indications at delivery include icteric amniotic fluid, large pale placenta, icterus of infant, pallor, hemorrhagic manifestations, edema, hepatosplenomegaly, athemia (hemoglobin less than 15 gm. and red blood count less than 5,000,000), positive direct Coombs test of infants' blood by heel puncture (this is more reliable than cord blood), and erythroblastosis, in excess of 20 per 100 white blood cells. Indications during neonatal period include increasing icterus, decline of hemoglobin and red blood cells, increase of erythroblasts, toxicity—listlessness, weakness, respiratory distress—and hemorrhagic manifestations.

Donor blood should preferably be same group as that of the infant, and Rh (d)-negative. Female donors are preferred. If group-specific blood is not available, one should use Group O, Rh (d)-negative blood. Fresh blood should be used if possible.

Heparin solution (1 cc. of 1,000 units per cc. in 100 cc. normal saline solution) should be used for rinsing syringes. Calcium gluconate is given to prevent tetany. Ten cubic centimeters of blood-group-specific substances A and B solution (Witebsky) should be added to donor blood if it is of group O.

MACDONALD, Pittsburgh.

TRYPTIC ACTIVITY OF THE STOOLS IN THE NEWBORN. D. O'BRIEN and B. W. POWELL, Great Ormond St. J. (No. 3) 33-35 (June) 1952.

The authors investigated the range of tryptic activity in the stools of the newborn, with the object of determining normal values. It was thought that such a study might aid in the early diagnosis of fibrocystic disease.

It was found that during the first two days low titers were generally obtained. Once the stool became fecal, titers below 1:50 were obtained only twice, and in all cases some degree of tryptic activity was present. In one case of meconium ileus there was no evidence of tryptic activity at two days.

Subjects tested at 2 days that showed a titer of less than 1:50 were retested at eight days, and in all except one titers of 1:100 or better were obtained. Those which had previously been tested at 3 to 10 days with resultant titers of 1:50 or less were retested at five weeks and three months and all had titers of 1:100 or better.

It is suggested that absence of tryptic activity even in the early days of life is supportive evidence for the diagnosis of fibrocystic disease when this diagnosis is suspected on clinical or familial grounds.

McGUIGAN, Evanston, Ill.

RH GROUPS AMONG EGYPTIANS. SALAH EL-DEWI, J. Roy. Egyptian M. A. **32**:883 (Nov.-Dec.) 1949.

Studies of the Rh factor in Egyptians showed an incidence of only 3% Rh-negative blood.

McGUIGAN, Evanston, Ill.

STUDIES ON THE SEX HORMONES DURING THE NEONATAL PERIOD. M. LELONG, M. F. JAYLE and P. BORNICHE, Arch. franç. pédiat. **4**:546, 1947.

The authors show that the newborn eliminates in the urine massive quantities of estrogens (estron, estradiol and estriol), comparatively twice as much as the mother in the same time. This elimination suddenly stops between the fourth and the seventh day. Elimination of pregnanediol, which is far lower than that of estrogen, is always of a shorter duration (two or three first days of life).

A new estrogenic body (to be identified) was found in the urine of the newborn (and was not present in the urine of the mother; this is slightly different from estron).

No gonadotropic hormone was found in the urine of the newborn.

MINKOWSKI, Paris, France.

OBSERVATIONS ON 150 ELECTROCARDIOGRAMS OF THE NEWBORN AND NURSING INFANT. G. LENZI, Clin. pediat. **29**:451 (Aug.) 1947.

A careful study of electrocardiograms taken on 150 healthy newborn and nursing infants is reported from the pediatric clinic at Bologna. Statistics are assembled as to the incidence and size of the different waves—P, Q, R, S, and T—of axis deviation, and of the time elements of the different phases of the heart beat. Among other data, it is reported that the T_3 wave was positive in 83.3% inverted in 1.7% and absent in 15% of the cases. The axis deviation was definitely to the right in 51%, slightly to the right in 39%, and in only one case to the left. The mean length of systole was 0.233 second and of diastole 0.174 second.

HIGGINS, Boston.

ANTISTREPTOLYSIN TITER IN THE BLOOD OF THE FETUS, THE NEWBORN INFANT, THE NEWBORN PREMATURE INFANT, AND THEIR MOTHERS, AND IN THE YOUNG INFANT. C. IMPERATO, Lattante **22**:641 (Nov.) 1951.

The antistreptolysin titer was determined by the method of Kalbak in blood of 4 fetuses, 17 premature infants, 50 newborn infants and their mothers, and also in the blood of 170 infants up to the age of one year.

The titer in the heart's blood of the fetus was less than 64 units and much lower than in the blood of their mothers; the titer in the blood from the umbilical cord of the premature newborn infant was from 28 to 640 units, about the same as that of their mothers; the titer in the blood from the umbilical cord of full-term newborn infants was from 28 to 1,800 units, about the same as or slightly higher than in the blood of their mothers.

The titer in the blood of infants falls during the first two months of life to a low figure, of about 20 units, but then may rapidly rise if there has been an infection.

HIGGINS, Boston.

FRACTURE OF THE CLAVICLE IN THE NEWBORN. CARLOS RAMIREZ BRAVO and ROBERTO GALECIO GOMEZ, Rev. chilena pediat. **18**:413 (June) 1947.

Following a review of the fractures of the clavicle found among newborn infants during the years 1945 and 1946 at the A. C. Sanhueza Maternity Hospital, the authors conclude that these fractures are more frequent among normal deliveries; that they are more frequent in those newborn with weight over the normal and that in a high percentage the fracture is total with fragment separation. The mechanism and treatment are discussed.

FROM THE AUTHORS' SUMMARY.

ASPHYXIA OF THE NEWBORN. HECTOR ARMAS GARCÍA, Rev. chilena de pediat. **18**:553 (Aug.) 1947.

This is a review of asphyxia of the newborn based upon a study of 711 cases of fetal distress encountered among 17,181 deliveries at the Maternidad del Salvador from 1943 to 1947. The author concludes that the most frequent causes of neonatal asphyxia are prolonged labor, exceptionally powerful uterine contracture, deliveries in elderly primiparas, placenta previa and operative trauma. Of the 711 patients 637 were born alive, and of these 302 presented asphyxia, while 335 were without asphyxia. Asphyxia was more frequent and more serious in premature than in full-term infants. The need for preventive measures is emphasized, and treatment is discussed.

KALISKI, San Antonio, Texas.

CAUSES FOR MORPHOLOGIC DIFFERENCES OF THE BLOOD OF MATURE AND IMMATURE NEWBORN INFANTS. F. KÜSTER, Ztschr. Kinderh. **65**:194, 1947.

The characteristics of the newborn blood—leukocytosis with shift to the left and increased numbers of erythroblasts, reticulocytes and erythrocytes—were previously explained on the basis of animal experiments by the effect of birth trauma and the formation of "hematopoietin,"

a hemopoiesis-stimulating substance. Experiments on newborn infants and the different behavior of premature infants do not substantiate this theory. Intrauterine lack of oxygen supply, on the other hand, does not explain why erythroblasts and reticulocytes are found in much lower numbers in the full-term infant than in the premature infant. Küster assumes that no immature cells are released into the blood by the bone marrow and that their origin is in the extramedullary hemopoietic centers, of which the liver is the most prominent. Studies of hepatic blood show that it contains more immature cells than does peripheral blood. Since the extramedullary blood-forming centers disappear during the latter part of fetal life, the smaller number of immature cells in infants born at the end of the fetal period is understandable. This also offers an explanation for the anemia of the premature infant, who is suddenly deprived of his extramedullary hemopoiesis at a time when the medullary blood formation has not yet fully taken over. The increased number of erythrocytes at and before birth may be caused by the low oxygen pressure in utero. A comparison with the high altitude polycythemia is acceptable only with reservation because of the difference between fetal and adult hemoglobin. There is disagreement in the literature as to whether or not the number of immature white cells is increased at birth. Küster does not attempt to answer this question but notes the independence of the red and white cell systems from each other. The increased number of leukocytes after birth is the only reaction which may be explained by birth trauma.

GLASER, Chicago.

THE PENICILLIN LEVEL AFTER PARENTERAL AND PERORAL APPLICATION IN NEWBORN AND IMMATURE BABIES. R. GOSTOVE and H. TROJANOVÁ, Pediat. listy **2**:245, 1947.

Rammelkamps' method was used for the estimation of the penicillin level of serum in 11 newborn children after intramuscular administration. The dose of 5,000 units given intramuscularly every three hours is considered the minimum, and it is necessary to increase the dose according to the need of the patient up to 8,000 units per kilogram in twenty-four hours. The levels after peroral and parenteral application of penicillin were followed in 9 immature infants. There is no difference between the two ways of administration. A good effect is gained from a dose of 5,000 units every four hours, given one-half hour before feeding, in a spoonful of sweetened tea.

FROM THE AUTHORS' SUMMARY.

ISO-IMMUNIZATION IN THE A B O SYSTEM AND MORBUS HAEMOLYTICUS NEONATORUM. STEN AXTRUP, Acta pædiat. **38**:45, 1949.

A description is given of some cases of hemolytic diseases of the newborn which may have been caused by an A B O incompatibility.

FROM THE AUTHOR'S SUMMARY.

SIGNIFICANCE OF THE RH GENOTYPE IN ERYTHROBLASTOSIS FETALIS. BIRGER BROMAN, Acta pædiat. **38**:50, 1949.

The author gives an account of recent advances in the serology of the Rh blood groups and of some typical cases of erythroblastosis fetalis. Many "deviations" from Levine's theory concerning the origin of erythroblastosis have proved to be only apparent, as is shown by the cases related.

FROM THE AUTHOR'S SUMMARY.

ICTERUS GRAVIS DUE TO ISOIMMUNIZATION WITH A-AGGLUTINOGEN. M. GROVE-RASMUSSEN, FOLKE TUDVAD, and GUNNAR JÖRGENSEN, Nord. med. **38**:1259 (June 25) 1948.

Two cases of icterus gravis are reported, in which the mothers had type O blood and the children type A. The maternal serum in both cases showed increased anti-A titer. This increase was greater in AB-medium than in saline medium, thus indicating immunization by A-agglutinogen. Both infants were given transfusions with compatible Rh-negative blood of group A. Transfusions with blood of group O not originating from the mothers are considered more adequate.

WALLGREN, Stockholm, Sweden.

TETANUS OF THE NEWBORN, TREATED WITH ANTITOXIN AND PENICILLIN. E. CHRYSSOCHERIS, Arch. Greek Paediat. Soc. **1**:97, 1947.

The case of a newborn infant in whom signs of tetanus developed on the seventh day is reported. Treatment with tetanus antitoxin and penicillin was instituted on the same day. A total of 200,000 units of antitoxin and of 800,000 units of penicillin was given intramuscularly over ten days. At the same time pentobarbital sodium and chloral hydrate were used as often as required. There was complete recovery.

DOXIADIS, Newcastle upon Tyne, England.

TETANUS NEONATORUM. J. H. DE HAAS, Maandschr. kindergeneesk. **17**:163, 1949.

Observation of two cases of tetanus neonatorum, one in which treatment was successful and one fulminant case, with an incubation period of seven and four days, respectively, leads to the following conclusions:

In the treatment of tetanus neonatorum sedatives, penicillin, and serum are complementary and, together with the most careful nursing, may be lifesaving. In this threefold combination serum is less dominant now than it was. Wound excision is no longer practiced. Sedatives are of utmost value.

Administration of phenobarbital in high dosage is strongly recommended; it is practically without risk of intoxication. In fulminant cases a stronger sedative will be necessary. Penicillin in high dosage influences the tetanus infection favorably and prevents complications. Oral administration of phenobarbital and penicillin prevents convulsions, eventually stimulated by repeated injections.

In the last decade tetanus neonatorum has become a curable illness instead of a nearly always lethal disease.

The complication of spondylolisthesis in tetanus neonatorum is mentioned (for the first time?). The posterior displacement of the second lumbar segment remitted spontaneously. It is possible that spondylolisthesis in tetanus neonatorum occurs as frequently as the compression fractures of the vertebral bodies in tetanus of older children. With the exactness of an experiment in both cases of tetanus neonatorum the vector of infection happened to be the umbilical scissors.

Recent literature suggests an increase of tetanus neonatorum in Holland in the past few years, after a long period of decreasing frequency. More acquaintance with the clinical aspect of tetanus neonatorum would show an apparently even higher incidence. The accurate and timely diagnosis is of utmost importance. Prevention of tetanus (neonatorum) is much more important even than successful treatment.

FROM THE AUTHOR'S SUMMARY.

MILD CASES OF ERYTHROBLASTOSIS FETALIS. S. I. DE VRIES, Nederl. tijdschr. geneesk. **91**:13 (Jan. 4) 1947.

To gain a better insight into erythroblastemia in erythroblastosis fetalis, de Vries tried to establish a differentiation between the normal and the pathologic blood picture in newborn infants. He investigated 85 normal children of Rh-positive mothers and found an average of 213 nucleated red blood cells per cubic millimeter. In 35 cases of clinically completely normal babies of Rh-negative mothers he found an average of 653 nucleated red blood cells per cubic millimeter. Besides this there existed a more toxic blood picture, with more young leucocytes, and there was anisocytosis more frequently. He concludes that these cases are cases of mild erythroblastosis fetalis and supposes that the frequency of this disease is probably much greater than has been assumed.

VAN CREVELD, Amsterdam, Netherlands.

Acute Contagious Diseases

NITROGEN AND POTASSIUM METABOLISM: THE REACTION PATTERN IN POLIOMYELITIS. A. G. BOWIE, F. M. MORGAN, and A. L. CHANEY, Am. J. M. Sc. **223**:532 (May) 1952.

Analysis of 17 cases of poliomyelitis with respiratory involvement indicated that the disease is divisible into an acute catabolic phase, a subacute catabolic phase, and an anabolic phase. During the acute catabolic phase there is urinary excretion of large quantities of nitrogen and

potassium, indicative of protoplasmic breakdown. These high levels persist longest in patients with the severest paralysis. In the subacute catabolic phase there is a lower excretion of potassium and nitrogen. The anabolic phase is characterized by synthesis of protein, with positive balance of potassium and nitrogen. The decrease in serum albumin during the anabolic phase may lead to shock in severe poliomyelitis. High serum potassium in the acute phase may lead to ventricular fibrillation, while low serum potassium accentuates the weakness.

BERLIN, Mount Vernon, N. Y. [A. M. A. ARCH. NEUROL. & PSYCHIAT.].

POLIOMYELITIS AND IMMUNIZATION AGAINST WHOOPING-COUGH AND DIPHTHERIA. H. STANLEY BANKS and A. J. BEALE, Brit. M. J. **2**:251 (July 29) 1950.

Banks and Beale made a study of the incidence of flaccid paralysis from poliomyelitis in relationship to the injection of materials for the immunization against whooping cough and diphtheria. The article is well summarized in the summary and conclusions as follows:

"Inquiry into the cases of poliomyelitis admitted to one London hospital during the years 1947, 1948, and 1949 showed a total of 111 paralytic cases of all ages admitted during the period. Of these, 14, all under 5 years old, had a paralysis of the limb which had received one or more of a course of immunizing injections within the previous two months. The interval between the last injection and the onset of paralysis in the majority of the cases was between 9 and 14 days. The paralysis followed combined pertussis and diphtheria (A. P. T.) prophylactic in nine cases, pertussis vaccine alone in one case, and diphtheria prophylactic A. P. T. alone in four cases. The paralysis, whether of arm or leg, conformed in general to a uniform pattern. It was not associated in time or place with any particular clinic or technique. It is concluded that during periods of high or moderately high prevalence of poliomyelitis there is a definite but probably small risk that inoculation with the type of whooping-cough and diphtheria prophylactic usually employed in this country will be followed by serious flaccid paralysis of the inoculated limb in young children. The paralysis particularly affects the muscles adjoining the site of inoculation."

BIRDSONG, Charlottesville, Va.

PREVENTION AND TREATMENT OF ORCHITIS PAROTIDEA. R. CROSNIER, Presse méd. **66**:1398 (Oct. 18) 1952.

It has been observed that orchitis, as a frequent complication of mumps, affects particularly the young adult and rarely affects the child who has not yet reached the puberty period. That is why the use of estrogens, which may bring back the testes to a prepuberal status, is efficient both in the curative and in the prophylactic treatment of orchitis parotidea. The author's experience enables him to state that this newer method is clearly superior to the usual routine management and even to infiltration anesthesia of the lumbar sympathetic.

The use of synthetic estrogens as a preventive measure should be started from the onset of the parotiditis and in the following dosage: diethylstilbestrol, 2 mg. daily for 10 days; hexestrol, 2 mg. daily for 10 days, and dienestrol, 1 mg. daily for 10 days.

The daily dose as curative is 5 mg. for diethylstilbestrol and hexestrol and 3 mg. for dienestrol for 10 days.

Orchitis parotidea correctly treated with estrogens does not leave any sequelae as a rule. Atrophy is exceptional; the testis preserves or resumes its tonicity and its normal volume.

FROM THE AUTHOR'S SUMMARY.

PROPHYLAXIS AGAINST MEASLES WITH GAMMA GLOBULIN IN FIFTEEN THOUSAND SUBJECTS, TREATED FREQUENTLY. LAURA MELE, Minerva pediat. **2**:591 (Dec.) 1950.

Fifteen thousand refugee children were given prophylactic doses of "anti-measles serum of Connaught, Toronto," with uncertain results, owing to the irregularity and insufficient dosage used in the treatments. More recent use of immune gamma globulin has given 99.86% absolute protection to 2,000 children.

HIGGINS, Boston.

FIRST CLINICAL RESULTS OF THE TREATMENT OF PERTUSSIS WITH STREPTOMYCIN AND AUREOMYCIN. ROLAND ROTT, Monatsschr. Kinderh. **99**:366 (Oct.) 1951.

Intramuscular administration of streptomycin in the dosage of 70 to 100 mg. per kilogram per 24 hours every six hours for 8 to 10 days was found to be effective in the treatment of uncomplicated pertussis; no toxic effects were observed. For cases complicated by pneumonia, addition of orally administered aureomycin in the dosage of 70 to 80 mg. per kilogram per 24 hours, given every six hours, and supplemented by intravenously administered aureomycin for extremely ill infants, was found to be more efficacious than streptomycin alone. Cerebral complications of pertussis, such as encephalopathy, were not influenced by either drug.

HARTENSTEIN, Medical Corps, United States Army.

TREATMENT OF PERTUSSIS WITH AUREOMYCIN. KLAUS SEELEMANN, Monatsschr. Kinderh. **99**:444 (Dec.) 1951.

Sixty-nine children with pertussis, half of them less than one year old, were treated orally with aureomycin in the dosage of 50 mg. per kilogram per 24 hours, given in four divided daily doses for 10 days. It was thought that one-third of the patients treated were not benefited. However, the drug was considered lifesaving for several seriously ill, small infants and in cases complicated by pneumonia.

HARTENSTEIN, Medical Corps, United States Army.

DIAPLACENTAL INFECTION OF THE FETUS WITH THE VIRUS OF GERMAN MEASLES DESPITE IMMUNITY OF THE MOTHER: ANALOGOUS OBSERVATIONS IN SMALLPOX. BÉLA SCHICK, Acta pædiat. **38**:563, 1949.

The problem of the transmission of rubella to the fetus of a mother immune to the disease is discussed. The occurrence of transmission is already established (Warkany). Analogous observations in smallpox have been published. The literature of the latter is reviewed. The consequence of such transmissions in different virus diseases constitutes a warning that a pregnant mother, even if she has had rubella in childhood or is herself immune to rubella without any previous history thereof, should not take care of her children or other patients suffering from rubella. The virus may invade the circulation of the mother and reach the fetus in spite of the immunity of the mother. The difference between cellular and humoral immunity of the mother is given as explanation of the infection of the fetus. Probably only humoral immunity of the mother would prevent the infection of the fetus. Cellular immunity would protect only the mother, not always the fetus. Even rapid (accelerated) reproduction of antibodies may come too late to prevent the passage of the virus through the placenta to the fetus.

FROM THE AUTHOR'S SUMMARY.

RUBELLA IN PREGNANCY AND CONGENITAL DEFORMITIES IN THE CHILD. M. ZEWI, Nord. med. **37**:416 (Feb. 27) 1948.

The author reports on two infants with malformations. In one of them there were double-sided congenital hydrophthalmos and clouded corneas. The infant's mother had suffered from rubella during the second month of pregnancy. In the second infant there were congenital bilateral cataract and microphthalmia. The infant's mother had had rubella during her first month of pregnancy. Both infants had low birth weight. They were retarded mentally and physically and had shown signs of congenital heart disease.

WALLGREN, Stockholm, Sweden.

REMOTE TROUBLE AFTER PAROTITIC MENINGOENCEPHALITIS. VERA OLDFELT, Nord. med. **40**:2200 (Nov. 26) 1948.

Of 75 patients with mumps meningoencephalitis 15 were found to be suffering from permanent effects at a follow-up study three to five years after the onset of the disease. One child suffered from total unilateral deafness, another child from severe epilepsy, and a third

displayed vestibular trouble. Several displayed diverse forms of eye and ear symptoms as well as of mental symptoms of a neurasthenic nature, both with and without headache. In one or two cases there was endogenous obesity. In a few cases changes were noted on the electroencephalograms.

WALLGREN, Stockholm, Sweden.

PREVENTION OF COMPLICATION IN SCARLET FEVER: TREATMENT WITH SULFANILAMIDE ("STREPTAN"). KJELL SIVERUD, Tidskr. norske lægefor. **68**:443 (Sept. 1) 1948.

Treatment with sulfanilamide during the first week of hospitalization did not reduce the incidence of secondary diseases. In one series 86 patients were treated and displayed 37 complications, while 39 patients with 7 complications were left without sulfonamide therapy. In a second series (172 cases) every second patient received sulfanilamide and the others were left as controls. There were 38 complications in the first group and 14 complications among the controls.

WALLGREN, Stockholm, Sweden.

MENINGOCOCCIC MENINGITIS IN CONNECTION WITH SMALLPOX VACCINATION. ARNE BORG, Tidskr. norske lægefor. **68**:543 (Oct. 15) 1948.

A full-term girl of 9 months was vaccinated against smallpox on Oct. 29, 1947. Nov. 6 she had fever and on the 12th day after vaccination her temperature rose to 40 C. (104 F.). There were no clinical signs of meningeal irritation. Spinal tap displayed purulent meningitis with meningococci. The patient was discharged healthy.

WALLGREN, Stockholm, Sweden.

TREATMENT OF SCARLET FEVER WITH PENICILLIN. TORBEN JERSILD, Ugesk. læger **110**:4 (Jan. 1) 1948.

Two thousand patients with scarlet fever at the Blegdam Hospital in Copenhagen were treated with 90,000 to 150,000 units of penicillin twice a day for six days. Then the patients were transferred to a convalescent, "clean," ward and discharged eight days, on the average, after admission. The streptococci disappeared from the nose and throat within 48 hours. No complications were observed either during the stay at the hospital or later.

WALLGREN, Stockholm, Sweden.

FURTHER EXPERIMENTS WITH DIPHTHERIA PHOSPHATE TOXOID. J. F. H. DE GRAAF, A. TASMAN and A. G. BRANDWIJK, Nederl. tijdschr. geneesk. **94**:2896 (Oct. 7) 1950.

Vaccination with diphtheria phosphate toxoid in 256 persons older than 12 years was only once followed by severe general and local reaction. Comparison with earlier investigations suggests that purification of diphtheria toxoid improves the possibility of using the toxoid in older persons. Among 30 subjects having a positive Schick reaction before the immunization, this reaction became negative after the first injection in 86.7% and in all of them after the second injection.

VAN CREVELD, Amsterdam, Netherlands.

Chronic Infectious Diseases

ISOLATION OF M. TUBERCULOSIS BY INOCULATION OF THE YOLK SAC OF EMBRYONATED EGGS. JOHN W. BRUECK and G. JOHN BUDDINGH, Proc. Soc. Exper. Biol. & Med. **80**:589 (Aug.-Sept.) 1952.

The yolk sacs of embryos of 5 to 8 days' preliminary incubation were inoculated through a small drill hole in the shell by means of 20-22 gauge needles. The material studied had not revealed the presence of tuberculous organisms by the usual methods employed. Six embryos were inoculated with each specimen. Beginning with the fourth day, the material from one embryo was transferred each day to a test tube, phenol was added, the material was shaken, allowed to stand two hours, and the fatty layer on the surface was removed to a slide and stained. Acid-fast bacilli were demonstrable in four to six weeks after inoculation in every instance in which positive results were obtained. Yolk sac cultures were proven to be positive for Mycobacterium tuberculosis by guinea pig inoculations.

HANSEN, Galveston, Texas.

STUDIES OF THE CEREBROSPINAL FLUID CIRCULATION IN TUBERCULOUS MENINGITIS IN CHILDREN: PART I. THE USE OF PENICILLIN AS A TRACER SUBSTANCE. JOHN LORBER, Arch. Dis. Childhood **25**:408 (Dec.) 1950.

An attempt was made to determine the optimum route of cerebrospinal administration of streptomycin in the treatment of tuberculous meningitis and to detect a block as soon as possible. The method employed was a modification of the Oxford technique of the use of penicillin as a tracer substance. It consists of the injection of a standard amount of penicillin into the cerebrospinal circulation at one point and its quantitative detection within 10 minutes from fluid obtained from a different site. This method was used on 55 occasions. It was found that in the absence of obstruction free and rapid mixing of the penicillin takes place irrespective of the site of its injection. Blocks were detected on 23 occasions, 18 of them within the spinal theca.

Determination of the lumbar protein content and of the lumbar-cisternal protein ratio as indices of spinal block was compared with the results of the penicillin assay. The former methods were found to be unreliable in cases of tuberculous meningitis.

Penicillin assay is not a suitable method for the detection of tentorial blocks or partial obstructions.

The greatest practical importance of penicillin assay is the detection of spinal blocks because appropriate treatment given in time will minimize its dangers. Spinal blocks are not necessarily permanent.

Indications for the performance of these tests are suggested. KELLY, Milwaukee.

CASE OF CEREBELLLAR TUBERCULOMA. DAVID LAWSON, Great Ormond St. J. (No. 3) 49 (June) 1952.

The case history of a 52-year-old man was reviewed. He had had a large tuberculoma of the cerebellum excised in 1905. Reexamination of the excised tissue confirmed the diagnosis. The patient after forty-seven years was in excellent health and showed no neurological disorder other than partial deafness. His is thought to be the first case of excision of an intracranial space-occupying lesion to meet with long-term success. MCGUIGAN, Evanston, Ill.

EFFICACY OF BCG VACCINATION: QUALITY AND DURATION OF THE POSTVACCINAL ALLERGY. M. FOURESTIER, G. BAISSETTE, L. BERGERON and A. GONZALES, Presse méd. **60**:325 (Mar. 5) 1952.

In Montreuil, an active program of BCG vaccination has been in progress for 28 months. Of a total population of 70,000, there are 17,600 under 20 years of age. The authors have divided these children into three groups.

The first group, comprising those under 5 years of age, number 4,100. In this group there have been 832 vaccinations (20.29%). The morbidity figure for primary infections, meanwhile, has fallen from 13.15% in 1948 to 8.96% in 1950.

In the second group, aged 5 to 15 years, there are 9,000, of whom 4,366 (48.51%) have received BCG. The morbidity figure for this group has declined from 9.71% in 1948 to 2.28% in 1950.

The third group, aged 15 to 20 years, number 4,500; of these, 270 (6%) have been vaccinated with BCG. In 1948 the morbidity figure for primary infection in this group was 1.1%, and in 1950 it declined to 0.6%.

Meanwhile, in a neighboring town where BCG vaccination was not practiced, the morbidity rate for the whole group rose from 6.03% in 1948 to 14.08% in 1950. The rate for the whole group in Montreuil declined from 5.55% in 1948 to 2.73% in 1950.

Morbidity figures for all other forms of tuberculosis also declined in Montreuil in this age group. The figures for persons over 20, as well as figures for neighboring towns, have risen in the meantime.

The skin reaction has become negative in one third of the vaccinated persons after two years. Whatever the reason for this, the authors recommended revaccination with BCG.

DAFFINEE, Melrose, Mass.

RELATIONS BETWEEN THE BLOOD FATTY ACIDS AND THE TUBERCULOUS CUTANEOUS TEST IN TUBERCULOUS CHILDREN. A. GIUA, Ann. ital. pediat. **3**:554 (Dec.) 1950.

The author has noticed the existence of a reaction between the saturation index of the hematic fatty acids and the intensity of cutaneous allergy to tuberculin in cases in which the smaller extension of the process and the quicker and more favorable course prove a greater resistance to the morbid agent. During the treatment with streptomycin the author has noticed a rise of the saturation index, particularly in the most favorable cases, with a tendency to an increase in the cutaneous reaction. He states that streptomycin does not have a cutaneous allergic action which can increase in loco the reactivity to tuberculin.

FROM THE AUTHOR'S SUMMARY.

TUBERCULOSIS IN CHILDREN. JULIO ESPINOZA, Rev. chilena pediat. **12**:551 (Dec.) 1950.

The author studied 3,009 necropsies made in the Children's Hospital Manuel Arriaran, in order to investigate tuberculous lesions. Conclusions are drawn concerning tuberculous morbidity and mortality in relation to foreign statistics. The primary tuberculous complex is analyzed in its pulmonary and extrapulmonary localization, its physical condition and perforations into thoracic organs with subsequent results. Pathogenesis of hematogenous dissemination (diffuse miliary tuberculosis, tuberculous meningitis, and organic tuberculosis) is discussed, and rates are given as to global frequency and age. Finally, tuberculous adenopathy of retrograde origin (cervical and abdominal form) is studied, starting from the primary pulmonary complex, and figures are given in relation to global percentages and age.

FROM THE AUTHOR'S SUMMARY.

TREATMENT OF INFANTILE TUBERCULOSIS. HOMERO SILVEIRA, Arq. pediat. **22**:7 (June) 1950.

The author, a tuberculosis specialist, reports his work in a clinic devoted to the treatment of childhood tuberculosis. "Gadusan" (colloidal copper morrhuate) is used as a therapeutic agent, and the report covers in detail five cases selected from extensive material. A regimen is first established for the management of childhood tuberculosis in its general aspects. The cases are then divided into two groups—the primary infection group and the reinfection group. After having made this classification, as well as the necessary subdivisions, the author comments on the clinical and roentgenological aspects characteristic of these infections. Diagnosis based on clinical, roentgenological and laboratory data is discussed. The author states his opinion regarding the treatment of childhood tuberculosis and, particularly, the value of "gadusan," which he holds rather high.

There are five case reports with illustrations of the x-ray findings.

CHRISTOPHERSON, Wilmette, Ill.

CLINICAL PICTURE OF TUBERCULOUS MENINGITIS TREATED WITH STREPTOMYCIN. PAUL BUNGER and WULF GEIGER, Deutsche med. Wchnschr. **75**:1579 (Nov. 24) 1950.

The more chronic course of tuberculous meningitis due to the treatment with streptomycin brought with it a prominence of symptoms previously regarded as atypical. On the basis of 106 cases, in adults and children, various particularly frequent syndromes are described. The most frequent syndromes are produced by vascular changes described as unspecific arteritis in chronic meningitis. The clinical picture depends on the localization of the lesions, which can also manifest themselves as chronic spinal meningitis. The preponderant lesions around the chiasma opticum and stalk of the hypophysis can produce ophthalmic and hypophyseal-diencephalic symptoms. Other symptoms such as an acute occlusion syndrome may be due to increased intracranial pressure; edema of the brain is of special importance. Furthermore, acute somatically conditioned psychoses are not very infrequent.

AMBERG, Rochester, Minn.

THE PENICILLIN TREATMENT OF EARLY CONGENITAL SYPHILIS. PETER PAUL, Deutsche med. Wchnschr. **75**:1688 (Dec. 15) 1950.

During the first trimenon 30 infants were admitted and 5 during the second one. Five infants were free from symptoms but were seropositive; the others presented severest specific symptoms. One or two penicillin treatments were given. Later, for safety's sake, two neoarsphenamine bismuth treatments were also given. Of three patients who died two were premature, dying on the first day of treatment. Clinically 32 infants were cured. Twenty-eight became seronegative, and three infants were seropositive, but the observation period was shorter than one-half year. One infant became seronegative after nine months.

AMBERG, Rochester, Minn.

PLASMA PROTEINS IN CHILDHOOD TUBERCULOSIS. EWERBECK, Monatsschr. Kinderh. **98**:150-151 (April) 1950.

By electrophoretic studies of plasma proteins in children with tuberculosis it was determined that albumin dropped while alpha globulin, especially the alpha$_1$ fraction, increased. The beta fraction increased less, and the gamma fraction remained stationary or fell. In severely ill patients this picture was more pronounced than in patients who were not so ill. With effective treatment and healing the values again returned to normal, the gamma-globulin fraction rising most rapidly. With aid of the electrophoretic diagram success or failure could be predicted shortly after therapy was initiated.

HARTENSTEIN, Madison, Wis.

CONTRIBUTION TO THE KNOWLEDGE OF HUMAN TOXOPLASMOSIS. HUGO JELKE, Svenska läk.-tidning **47**:1 (Jan. 5) 1950.

The author reports on three cases of toxoplasmosis. 1. A boy of 8 years had hemiplegia, epileptiform fits, mental deficiency, chorioretinitis, and intracranial calcifications. 2. A boy 3 weeks old had hydrocephalus, signs of encephalitis, microphthalmos, chorioretinitis, intracranial calcifications, and xanthochromic, protein-rich spinal fluid. The patient died two weeks later. There were pathologically typical signs of toxoplasmosis. 3. A girl at 2 months of age suffered from long-standing jaundice, hepatomegaly and splenomegaly, and occult bleeding from the bowel. Two months later strabismus was noted. When she was 1½ years of age chorioretinitis and intracranial calcifications were demonstrated. In all three cases dye tests, complement-fixation tests and toxoplasmin tests were positive for mother and child.

WALLGREN, Stockholm.

GLANDULAR TYPE OF TOXOPLASMOSIS IN PREGNANCY. SVEN GARD and HENNING MAGNUSSON, Svenska läk.-tidning **47**:2141 (Sept. 29) 1950.

A case is reported of toxoplasmosis in a 23-year-old woman, manifested in the second week of her first pregnancy in the form of fever of short duration and glandular swelling in the neck. Biopsy of a gland showed inflammatory process of the type characteristic of toxoplasmosis, but no evident protozoas were found. There were strongly positive reactions in increasing intensity to a dye test and a complement-fixation test. The infant was full term and healthy.

WALLGREN, Stockholm.

Diseases of Blood, Heart and Blood Vessels, and Spleen

APICAL DIASTOLIC MURMURS IN CONGENITAL HEART DISEASE: THE RARITY OF LUTEMBACHER'S SYNDROME. A. S. NADAS and M. M. ALIMURUNG, Am. Heart J. **43**:691 (May) 1952.

Apical diastolic murmurs were found in 19 per cent of 100 consecutive patients with noncyanotic congenital heart disease. Twenty patients with noncyanotic congenital heart disease and apical diastolic murmurs were studied extensively to determine the accurate anatomic diagnosis. Atrial septal defect, ventricular septal defect, and patent ductus arteriosus were the

commonest lesions found in this series. There was no example of Lutembacher's syndrome in this group of patients. Twenty-five thousand consecutive autopsies studied contained only five cases of Lutembacher's syndrome—representing 6% of all atrial septal defects.

Physiologic mechanisms explaining the origin of these "functional mitral murmurs" in congenital heart disease are suggested. Diagnostic criteria differentiating the "organic" mitral murmur from the "functional" ones are pointed out. The probability of the development of mitral stenosis in patients with atrial defect is so small that an operation aimed at closure of the defect is highly desirable.

FROM THE AUTHORS' SUMMARY.

VARIATIONS IN BLOOD PRESSURE IN HOSPITALIZED CHILDREN. GEORGE W. CLAYTON and JAMES G. HUGHES, J. Pediat. **40**:462 (April) 1952.

George Clayton and James Hughes remind us that emotions affect children's blood pressures, too. In the hospitalized child, repeated blood pressure readings should be obtained for proper evaluation. In general, acutely ill children have a more stable blood pressure pattern.

MACDONALD, Pittsburgh.

HEMOGLOBIN LEVELS IN INDIANS. J. MILLAR, Canad. M. A. J. **67**:417 (Nov.) 1952.

Studies of hemoglobin levels in Indians showed an alarming degree of anemia in the younger children. This anemia was most marked between 6 months and 2 years, the lowest level being at 1½ years, when the average reading was 56.6%. At this age level 84.5% of children examined were anemic. The percentage rose steadily to age 6, when it was 11.2%. The anemia is on a nutritional basis.

McGUIGAN, Evanston, Ill.

APPARENT IDIOPATHIC HEINZ BODY ANEMIA. I. A. B. CATHIE, Great Ormond St. J. (No. 3) 43 (June) 1952.

The author presents a case of apparent congenital hemolytic anemia associated with a persistently high incidence of Heinz bodies in the red blood cells Careful taking of the history failed to reveal the ingestion of any drug or poison, and the Heinz bodies appeared to be a developmental rather than a toxic phenomenon.

McGUIGAN, Evanston, Ill.

CLINICAL PICTURE OF SPLENIC AND PORTAL VEIN STENOSIS IN CHILDHOOD. RUDOLFA WYSS, Helvet. paediat. acta **7**:452 (Nov.) 1952.

Six cases of splenic and portal vein stenosis which were observed at the Kinderspital Zurich and the records of which were carefully examined after release from the hospital are described with consideration of the literature. The patients ranged in age from 18 months to 12 years. All showed a tremendous enlargement of the spleen, anemia, and leukopenia and in all but two there was a hematemesis. In four patients splenectomy was performed, and two of these died shortly after the operation. The other two showed a definite improvement of their prognosis so far as life is concerned. At autopsy, two had a questionable and another a definite stenosis of the portal vein. Of the two on whom no splenectomy was performed, one died from loss of blood after four years. The other is living, and 30 years have passed since the condition was diagnosed and 15 years since the last bleeding.

The splenic and portal vein stenosis is a symptom complex caused by congestion in the area drained by the portal vein. The cause of the congestion cannot be determined clinically or in many cases at necropsy. The disease runs a chronic course, the danger lying in death by hemorrhage. There is no known conservative treatment. The operative therapy of splenectomy attempts to give partial relief to the portal circulation and to remove the splenogenic bone marrow inhibition. The postoperative prognosis for the cases of splenic vein stenosis is much better than that for the portal vein stenosis, in which one must reckon with recurring hemorrhage even after the operation. It is impossible to make a differential diagnosis between splenic and portal vein stenosis clinically. The prognosis is generally poor in cases in which there is no

operation; however, there are exceptions. Anatomicopathologic splenic findings are those of a congestive spleen, and in later stages one may also find fibroadenia. A differential diagnosis with Banti's syndrome is in certain cases neither clinically nor anatomicopathologically possible.

FROM THE AUTHOR'S SUMMARY.

TREATMENT OF CHRONIC MYELOGENOUS LEUKEMIA OF CHILDHOOD WITH URETHANE. HUGO ALTHOFF, Monatsschr. Kinderh. **99**:441 (Dec.) 1951.

X-ray therapy of chronic myelogenous leukemia in children was found to be preferable to treatment with urethan.

HARTENSTEIN, Medical Corps, United States Army.

ARTERIOVENOUS FISTULA IN THE PULMONARY CIRCULATION. ADA MIDDELHOVEN, Maandschr. Kindergeneesk. **20**:157 (June) 1952.

The author reports the case history of a 5-year-old "blue child," presenting the classical symptoms due to an arteriovenous fistula in the pulmonary circulation; she was successfully operated on. The fact is stressed that this condition can be diagnosed with certainty by accurate physical examination and simple x-ray procedures, and that such diagnosis may be life-saving.

In contrast to the well-defined clinical picture, little is known about the anatomical origin of this condition; it seems likely that different types of pathology may give rise to the same hemodynamic disorder.

FROM THE AUTHOR'S SUMMARY.

Diseases of Nose, Throat, and Ear

LARYNGEAL PAPILLOMATA IN CHILDREN. D. S. GARRELL, Canad. M. A. J. **67**:425 (Nov.) 1952.

A case of multiple laryngeal papillomata in a 2-year-old child is reported. The literature is reviewed, and methods of treatment are discussed.

MCGUIGAN, Evanston, Ill.

PATHOGENESIS OF HYPOPHARYNGEAL DIVERTICULUM WITH SPECIAL REFERENCE TO HEREDITY. HARRY BJÖRK, Acta oto-laryng. **42**:202 (June) 1952.

Björk reports the occurrence of diverticulum of the hypopharynx in three sisters and the mother of the siblings. This relationship in itself is strong evidence that hereditary factors play an important part in this condition. The histories of these patients were analyzed, as were the x-ray studies and the postoperative course. Reasoning from these factors, Björk believes that abnormal obstruction at the mouth of the esophagus is not a prerequisite for diverticulum formation. He states that if the posterior wall of he hypopharynx is weak, a pressure of deglutition of physiologic proportion is sufficient to cause the formation of a pouch.

LEWY, Chicago [A. M. A. ARCH. Otolaryng.].

Diseases of Lungs, Pleura, and Mediastinum

ADRENOCORTICOTROPIC HORMONE IN THE CONTROL OF ACUTE INFANTILE BRONCHIOLITIS. STUART H. WALKER, J. Pediat. **41**:528 (Nov.) 1952.

In severe cases of acute infantile bronchiolitis, corticotropin appears to reduce the exudate, the edema, and vascularity of the bronchiolar wall, thereby restoring alveolar ventilation. The original exudate is not affected.

MACDONALD, Pittsburgh.

HEMOPTYSIS IN AN INFANT. P. GIRAUD, A. BERNARD, H. MÉTRAS, T. MANGIAPIN, M. LABITTE and L. HARTUNG, Presse méd. **59**:1787 (Dec. 25) 1951.

Hemoptysis, a rare symptom in young infants, was observed in a boy of six months. Roentgenological studies with bronchography showed a large congenital cyst of the left inferior lobe. Lobectomy was successfully performed.

DAFFINEE, Melrose, Mass.

CASE OF IDIOPATHIC PULMONARY HEMOSIDEROSIS CURED BY SPLENECTOMY. M. CORDEIRO, Helvet. paediat. acta **7**:501 (Nov.) 1952.

A further case of idiopathic pulmonary hemosiderosis healed by splenectomy is described.

FROM THE AUTHOR'S SUMMARY.

Diseases of the Gastrointestinal Tract, Liver, and Peritoneum

URINARY 17-KETOSTEROIDS IN CHRONIC LIVER DISEASE. T. L. WILLIAMS, A. CANTAROW, K. E. PASCHKIS and W. P. HAVENS JR., Endocrinology **48**:651 (June) 1951.

Administration of testosterone to normal and eunuchoid human subjects has been shown to be followed by an increase in urinary 17-ketosteroids. It has also been reported that urinary 17-ketosteroid values are rather consistently low in patients with hepatic disease.

Experiments here reported indicate that the daily urinary excretion of total neutral 17-ketosteroids is considerably lower in patients with chronic liver disease than in normal subjects, and suggest that the capacity of the organism (presumably of the liver) for conjugation of metabolites of testosterone is impaired in subjects with chronic liver disease.

JACOBSEN, Buffalo, N. Y.

EFFECT OF VARIOUS HORMONES UPON THE HEPATIC SYNTHESIS OF CHOLESTEROL IN RATS. RAY H. ROSENMAN, MEYER FRIEDMAN, and SANFORD O. BYERS, Endocrinology **51**:142 (Aug.) 1952.

The biliary concentration of cholesterol previously was shown to be an indicator of hepatic synthesis of cholesterol in rats. This method was used to study the influence of corticotropin, adrenocortical steroids, and the sex hormones. The results suggest that hepatic synthesis of cholesterol in rats is depressed by estrogenic hormone and augmented by administration of corticotropin.

JACOBSEN, Buffalo.

EPIDEMIC DIARRHEA IN A SCHOOL FOR BOYS. THEODORE H. INGALLS and SIDNEY A. BRITTEN, J. A. M. A. **146**:710 (June 23) 1951.

Epidemic diarrhea in a boarding school is reported. The conclusion was reached that the diarrhea was spread by personal contact, since the pattern of spread through the population denoted personal contact rather than eating the same food. When an epidemic is abrupt in appearance and of short duration the arrangement of the cases by four-hourly intervals illustrates the pattern of spread. Search for ambulatory patients indicates the extent and severity of the epidemic.

KIMBALL, Evanston, Ill.

EFFECTS OF DIET, PARTIAL HEPATECTOMY AND GROWTH-PROMOTING FACTORS ON THE COMPOSITION OF THE RAT LIVER. MARY E. DUMM, ELAINE P. RALLI, HERBERT GERSHBERG and BERTRAM LAKEN, J. Nutrition **47**:11 (May 10) 1952.

Dumm and her colleagues studied the effect of normal diets (22% protein) and low-protein, high-fat diets (9% protein) on the liver constituents of intact rats. The increase in liver lipids was associated with a decrease in the nitrogen content of the liver. The injection of liver extract was associated with a decrease in the liver lipids, but not as consistent a decrease as that occurring with vitamin B_{12}. Increasing the protein content of the diet to 16% after partial hepatectomy was associated with some lipotropic effect and an increase in the liver nitrogen, but this was not as uniform as when the protein in the diet was increased to 26%. The administration of both liver extract and vitamin B_{12} was associated with microscopic evidence of regeneration of liver cells, more marked in the animals receiving vitamin B_{12}. In the animals changed to the 26% protein diet, regeneration of the liver cells was present but to a less marked degree.

FREDEEN, Kansas City, Mo.

HEPATIC NECROSIS INDUCED BY DIETARY MEANS. JOHN R. MCLEAN and J. M. R. BEVERIDGE, J. Nutrition **47**:41 (May 10) 1952.

J. R. McLean and J. M. R. Beveridge concluded that some factor other than the sulfur-containing amino acids and alpha-tocopherol has a profound effect on the development of acute massive hepatic necrosis in rats. Increasing levels of fat produced concomitant increases in the necrogenicity of the basal diet. A ration free from fat prevented the development of liver necrosis. Variation in the carbohydrate level had no discernible effect on the necrogenicity of the basal diet.

FREDEEN, Kansas City, Mo.

OBSERVATIONS ON THE TREATMENT IN A CASE OF GLYCOGEN STORAGE DISEASE. GEORGE H. LOWERY and JAMES L. WILSON, J. Pediat. **35**:702 (Dec.) 1949.

The patient, a 2-year-old boy with von Gierke's disease, has been carried on a high-protein, low-carbohydrate diet. On this type of diet there has been less fluctuation of blood sugar and less rapid fall to hypoglycemic values. Hypoglycemic convulsions have been averted, but the fundamental disease state has persisted.

MACDONALD, Pittsburgh.

CELIAC DISEASE TREATED WITH POLYOXYETHYLENE SORBITAN MONOOLEATE. HOWARD BOYD and FRANCIS HELFRICK, J. Pediat. **38**:493 (April) 1951.

Polyoxyethylene sorbitan monooleate is a detergent that diminishes the size of fat globules by lowering their surface tension. Boyd and Helfrick gave it to two patients with celiac disease. The dose was 40 mg. to each gram of ingested fat. The patients given this material in this manner were able to eat a normal diet without remission of the disease.

MACDONALD, Pittsburgh.

USE OF CAROB FLOUR (AROBON) IN A CONTROLLED SERIES OF INFANT DIARRHEA. THOMAS R. PLOWRIGHT, J. Pediat. **39**:16 (July) 1951.

The "locust and wild honey" which St. John the Baptist found in the wilderness was apparently the fruit of the carob tree. The carob is a dark evergreen tree that grows wild in Mediterranean countries; it is sometimes grown in the southern United States for its shade. The pulp of the fruit of this tree is usually fed to cattle but is sometimes eaten by men. In Mediterranean countries this fruit is often used as food by the poorer people.

In Barcelona, during the Spanish Civil War, Ramos noticed that the poorer-class children of the city who ate the carob fruit had fewer diarrheal diseases than children of the wealthier classes. With this in mind, he concocted a mixture of carob fruit and starch for the treatment of diarrhea. Under the name of "Arobon," this concoction has been used, with some success, in the treatment of diarrhea in Europe. "Arobon" is now available in the United States.

Plowright used "Arobon" in 20 cases of infantile diarrhea with an equal number of controls. All patients given "Arobon" recovered in about half the time of the controls. No adverse side effects were noted.

MACDONALD, Pittsburgh.

INVESTIGATIONS ON THE BACTERIAL FLORA, pH, AND SUGAR CONTENT IN THE INTESTINAL TRACT OF INFANTS. GIULIO J. BARBERO, GRETCHEN RUNGE and DOANE FISCHER, J. Pediat. **40**:152 (Feb.) 1952.

The authors report on the bacterial flora and the intestinal pH of infants fed either human or cow's milk. In general, the duodenum was relatively sterile. Bacteria, mainly enterococci, were found in the jejunum and in increasing numbers toward the jejunum. In the cecum and distal to it there was marked bacterial proliferation. Intestinal pH reached a peak of 7.0 to 8.0 in the cecum and tended to fall in the distal portion of the colon. This tendency toward an acid level was greater in breast-fed infants than in those fed cow's milk. Varying amounts of reducing sugar appeared throughout the entire length of the intestine in all infants, no matter what the type of feeding was. Average pH in breast-fed infants was 5.5; in infants fed evaporated milk and maltose-dextrin it was 6.7.

Flora of the breast-fed infant's stools was comprised of over 90% Gram-positive bacilli. The stool flora varied in infants fed cow's milk. None of the available brands of "adapted" milk produced stools with acidity and bacterial flora similar to those of breast-fed infants.

MACDONALD, Pittsburgh.

GASTRIC RESPONSE OF NORMAL CHILDREN TO URECHOLINE. J. ALBRIGHT JONES, BERNARD MARGOLIS and THOMAS POTTERFIELD, J. Pediat. **40**:456 (April) 1952.

Jones, Margolis, and Potterfield thought that bethanechol chloride might facilitate the passage of a tube through the pylorus in children difficult to intubate. The result was that the drug increased apprehension and nervousness in the children studied. There was also marked increase in free and total stomach acid levels. The authors conclude that the drug is unsuitable for use in facilitating the passage of a tube through the pylorus during duodenal intubation.

MACDONALD, Pittsburgh.

PATHOLOGY OF EXPERIMENTALLY PRODUCED LYE BURNS AND STRICTURES OF THE ESOPHAGUS. L. H. BOSHER JR., T. H. BURFORD, and LAUREN ACKERMAN, J. Thoracic Surg. **21**:483 (May) 1951.

The authors studied lye burns of the esophagus in dogs by the following method. Ten to 15 cc. of a 10% sodium hydroxide solution was introduced into the esophagus of dogs. The lower esophagus was temporarily occluded to prevent the lye solution from entering the stomach. The solution was allowed to remain in the esophagus for 60 seconds, and then removed by suction.

Nine of the 14 dogs studied died from 1 to 12 days after the burn of esophageal perforation, pneumonia, or laryngeal edema, or were killed. The remaining five animals were maintained from 4 to 18 weeks.

Within 24 hours of the time of the burn epithelial destruction, edema, and congestion were evident, especially marked in the lower half of the esophagus. It was then observed that the submucosa and muscularis were heavily infiltrated with inflammatory cells. Arterial and venous thrombosis, especially prominent in the submucosa, was observed. Liquefaction of the mucosa and submucosa occurred by the fifth day. There was evidence of secondary bacterial invasion and muscle necrosis.

Reepithelization of the esophagus under these conditions required about six weeks. By this time stricture formation was evident.

In view of the pathological changes observed in these experiments, the authors believe that early dilatation in cases of lye burn of the esophagus is illogical and is harmful to the normal healing processes.

MEYER, Chicago.

GALACTOSEMIA AND ITS RELATION TO LAENNEC'S CIRRHOSIS: REVIEW OF THE LITERATURE AND PRESENTATION OF SIX ADDITIONAL CASES. EDWARD H. TOWNSEND JR., HOWARD H. MASON, and PAUL S. STRONG, Pediatrics **7**:760 (June) 1951.

Galactosemia is a specific metabolic deficiency capable of profound effects on growth, development, and well-being. The essential features of the illness are feeding difficulties dating to the neonatal period, malnutrition, hepatomegaly, galactosuria, mental deficiency and not infrequently nuclear cataracts. The authors summarize 9 cases from the literature and report 6 additional cases, bringing the total number of reported cases to 15. Undoubtedly this represents a small minority of the total number of cases that have existed.

The authors report one case in detail. An infant 6 months of age presented the clinical picture described above and, in addition, physical and physiologic evidence of impaired liver function. These signs gradually disappeared on a galactose-free regimen. An inadvertent break in this dietary regimen caused a reappearance of all symptoms, and a liver biopsy performed at this time showed Laennec's cirrhosis.

Then the autopsy protocols of all infants who had died of Laennec's cirrhosis were studied and an additional case was discovered in which galactosemia had played an etiologic role.

Galactosemia produces liver dysfunction through a combination of hypergalactosemic blood levels which are of themselves injurious and the hypoglucosemic levels induced by the high blood sugar level caused by the hypergalactosemia. With proper dietary management these infants may grow normally to maturity, at which age they can consume normal quantities of milk and milk products without obvious physical or physiologic damage. The authors emphasized the frequency of mental retardation as a component of the syndrome.

FROM THE AUTHORS' SUMMARY.

SOURCE OF ERROR IN TEST FOR STOOL TRYPSIN. P. C. GAFFNEY, Pediatrics **8**:82 (July) 1951.

A source of error in the gelatin film test for stool trypsin is reported. Identical results are obtained with kodak® and Dupont film when incubation at room temperature for two hours is used.

The gelatin on Dupont film incubated at 37 C. for one hour is digested by stools from infants with mucoviscidosis and also by distilled water, as well as by stools from healthy infants. Stools from some infants with mucoviscidosis will digest the gelatin on kodak® film when incubated at 37 C. for two hours.

Kodak® film should be used if the preparations are to be incubated at 37 C. for one hour, as is recommended in the original description of the technique.

FROM THE AUTHOR'S SUMMARY.

PYLORIC STENOSIS IN FOUR FIRST COUSINS. C. O. CARTER and T. R. SAVAGE, Arch. Dis. Childhood **26**:51 (Feb.) 1951.

There is evidence that genetic factors play some part in the causation of pyloric stenosis. Identical twins appear to be more often both affected than fraternal twins of like sex, and the incidence in brothers and sisters of children with pyloric stenosis is higher than in the general population.

A family of 11 brothers and sisters is presented, 5 of whom have had children, and of these 4 have had a child with pyloric stenosis in which the diagnosis was confirmed at operation.

This family provides additional evidence that genetic factors are in part responsible for the condition. Also it suggests that if a recessive gene of incomplete manifestation is at work then a high proportion of the population must be carriers.

KELLY, Milwaukee.

ENTEROGENOUS CYST CAUSING CONGENITAL INTESTINAL OBSTRUCTION. E. W. PARRY, Arch. Dis. Childhood **26**:63 (Feb.) 1951.

A case of congenital intestinal obstruction due to intrauterine volvulus of an enterogenous cyst in the jejunum, with a second large cyst in the lower ileum, has been described. No other record of a cyst causing congenital obstruction has been found in the literature.

KELLY, Milwaukee.

BILE PIGMENTS IN THE STOOLS OF INFANTS. S. D. V. WELLER, Arch. Dis. Childhood **26**:88 (Feb.) 1951.

Over 160 stools were tested from 102 infants under 4 months of age without obstructive jaundice. In 20% of babies stercobilin was not detected. It is therefore necessary to test for both bilirubin and stercobilin to prove biliary obstruction in children of this age group.

KELLY, Milwaukee.

QUADRIPLEGIA COMPLICATING GASTROENTERITIS. BERNARD SCHLESINGER and R. G. WELCH, Great Ormond St. J. (No. 3) 14 (June) 1952.

In five patients with severe gastroenteritis quadriparesis and mental impairment developed. All were severely dehydrated. No definite cause for the complication was ascertained, but it is believed that there was probably a partial cavernous sinus thrombosis.

MCGUIGAN, Evanston, Ill.

LABORATORY TESTS IN THE DIAGNOSIS OF CELIAC AND FIBROCYSTIC DISEASE. W. W. PAYNE, Great Ormond St. J. (No. 3) 23 (June) 1952.

The diagnosis of celiac disease and its differentiation from fibrocystic disease may be very difficult and calls for extensive laboratory investigations which must be carefully correlated. A depressed fat absorption by itself is not sufficient evidence of celiac disease and requires, in the absence of a clearcut clinical picture, a sugar-tolerance curve and chylomicron count. In fibrocystic disease, the most valuable test is that of tryptic activity of the duodenal juice. Also helpful are the tryptic activity of the stool and the amino acid curve. McGUIGAN, Evanston, Ill.

CASE OF CARCINOMA OF COLON. G. H. NEWNS and CHARLES DONALD, Great Ormond St. J. (No. 3) 64 (June) 1952.

A case of carcinoma of the colon in a 10½-year-old boy is reported. Eighteen months later there was no evidence of recurrence. McGUIGAN, Evanston, Ill.

SINUSITIS AND ASSOCIATED LABYRINTHITIS. J. S. McKIM, Great Ormond St. J. (No. 3) 81 (June) 1952.

In an 11-year-old girl with acute pansinusitis marked signs of labyrinthitis disturbance developed. No otologic pathologic changes were present. Treatment with antibiotics and central lavage resulted in prompt recovery. McGUIGAN, Evanston, Ill.

CIRRHOSIS OF THE LIVER IN CHILDREN. G. COELHO, Indian J. Child Health **1**:455 (Sept.) 1952.

A general discussion of the problems of cirrhosis of the liver is presented. Incidence, diagnosis, prognosis, and treatment are briefly considered. McGUIGAN, Evanston, Ill.

ABDOMINAL TUMORS IN INFANCY. MARCEL FÈVRE, Presse méd. **59**:109 (Jan. 27) 1951.

Fèvre enumerates the various types of tumor found in the abdomen in early life and stresses the fact that early diagnosis and surgical removal are essential. DAFFINEE, Melrose, Mass.

RESPIRATORY DISTURBANCES IN CHILDREN SUFFERING FROM TOXICOSIS. C. CHOREMIS and D. MELLISAKIS, Ann. paediat. **176**:379 (June) 1951.

The study of the respiratory disturbance in toxicosis, which is one of the pathognomonic symptoms of this condition, proves that the disturbance observed is not of the Kussmaul type as it was previously generally accepted; that this respiratory disturbance appears under two main types, (*a*) the polypneic, of peripheral origin, and (*b*) the paralytic, of central origin. The second type is more frequent and severer; infants with this type usually die. Of course the difference in frequency between the first and the second type is also due to the fact that the infants are brought to the hospital in an advanced stage of the disease during which the second type predominates.

The distinction into two types of breathing is in accordance with the authors' conception of the pathogenesis of toxicosis. They believe that the pathophysiological course of the typical toxicosis runs two phases, (*a*) physiochemical (sudden loss of fluids and minerals), and (*b*) chemotoxic, during which there is an absorption from the gastrointestinal tract of products of bacterial decomposition and of severe alteration of the digestive function as well. The absorption of these substances leads to a profound disturbance of the metabolism of the body and finally results in coma. FROM THE AUTHORS' SUMMARY.

CEREBROSPINAL FLUID CHANGES IN TOXICOSIS DUE TO INFANTILE DIARRHEA. C. CHOREMIS; B. KONSTANTINIDES; M. KIZA; A. MARKIDOU, and M. NESTORIDOU, Ann. paediat. **177**:77 (Aug.) 1951.

The authors point out that cerebrospinal fluid changes in toxicosis should be explained as a result of the biochemical changes of the blood and that consequently these alterations are limited to those elements of the cerebrospinal fluid which depend upon the biochemical state of the blood.

The increased permeability of the membranes in infancy permits the passage of various constituents of the blood, mainly sugar and chlorides and in a very small degree of potassium and phosphorus, in accordance with Donnan's equilibrium.

The alkali reserve is constantly decreased after the reduction of the blood alkali reserve; the decrease should be attributed to the low blood sodium in toxicosis. In the support of the above view is the fact that the blood changes following the infusion of different mineral solutions are accompanied in some way by similar cerebrospinal fluid changes.

FROM THE AUTHORS' SUMMARY.

ROENTGENOLOGIC FACTOR IN INTERMITTENT GASTRIC VOLVULUS IN INFANCY. R. DELLA CASA, Arch. ital. pediat. e puericolt. **13**:217, 1949.

In seven infants (one free of symptoms and six with frequent vomiting) the roentgenologic examination showed a particular torsion of the stomach here described.

In two of the children repeated roentgenologic investigation confirmed the diagnosis of intermittent mesenterial-axial volvulus; in another, who could not be examined again, the roentgenogram shows a deformity of the stomach similar to the one seen in the first two cases. The colon, the ileum, and the stomach in all the cases are filled with air.

In the second case the shape and site of the stomach underwent progressive change while under observation, showing a torsion on the transverse axis; the fundus is thus the most dependent part, while the body and the antrum, distended by gas, appear situated beneath the diaphragm. In the other cases this deformity was seen on the first examination; in the first case a second roentgenologic investigation, made 20 days after the first, failed to show the previously observed change.

The author feels that a systematic roentgenological investigation in infants, especially those suffering from gastric troubles (regurgitations and vomiting) will, rather frequently, disclose gastric deformities of various degree. In effect, in children are often seen those predisposing factors which many authors consider are most important for bringing about volvulus—colon or stomach filled with air and hyperperistalsis. A defective fixation of the organ, especially laxity of the small omentum and the gastric-splenic ligament, must also be taken into consideration.

In the first case, in spite of the scanty clinical symptoms, the early frequent food vomiting led the author to recognize the gastric deformity as the cause of the troubles.

The author concludes by advocating a more frequent use of roentgenologic investigation in all those cases in which repeated vomiting is seen together with regular stools.

While removing, if possible, all these pathogenic factors which are liable to bring about the gastric deformity, the physician must devote his attention to the treatment of the vomiting.

FROM THE AUTHOR'S SUMMARY.

ACUTE INTUSSUSCEPTION IN THE INFANT AND CHILD. V. ZAFFAGNINI and G. NIERO, Clin. pediat. **31**:321 (June) 1949.

An analysis is given based on 35 cases of intussusception in children seen in the Surgical Clinic at Bologna over a period of 10 years; 68% were in infants under one year of age, a majority of these being between 5 and 9 months old; 77% were in male children, and 89% were ileocecal. The severity of the symptoms was not related to the length of the tumor. In 2 cases, out of 8 in which the attempt was made, intussusception was reduced by barium enema. Operative treatment was used in the other 33 cases. Appendectomy was deemed necessary in 10 of the cases because of vascular thrombosis and threatened gangrene. All the patients operated upon on the first day after onset recovered.

HIGGINS, Boston.

QUINCKE'S EDEMA AND INTESTINAL INTUSSUSCEPTION. P. MARINELLI, Clin. pediat. **31**:355 (June) 1949.

A 6-year-old boy had 4 attacks of angioneurotic edema, each with edema in a different part of the body—ankle, lip, scrotum, and sacrum—but all accompanied by abdominal pain. After the last attack there were vomiting and bloody stools. At operation an ileal intussusception, 20 cm. long and 60 cm. from the ileocecal valve, was found; it could not be reduced and had to be corrected by resection and an anteroenteral anastomosis. Recovery was uneventful; the attacks

of edema did not recur. The occurrence of localized edema with spasm of the intestinal muscle would seem to be the cause of the intussusception in this case and perhaps in other so-called idiopathic intussusceptions and in the intussusceptions seen in the course of Schoenlein's purpura.

HIGGINS, Boston.

AMEBIC HEPATITIS IN CHILDREN. E. SORAGNI, Clin. pediat. **31**:414 (July) 1949.

In two children in Modena amebic dysentery with hepatitis developed; one of the children had an amebic abscess of the liver. Amebiasis had not been endemic in this province previous to the recent war, but now its presence must be ruled out in cases of dysentery.

HIGGINS, Boston.

VON GIERKE'S DISEASE. T. PORCELLI, Pediatria **57**:817 (Nov.) 1949.

Von Gierke's disease is discussed at some length (65 pages), and the case of a boy of 18 months with the glycogen disease is reported. The liver was enlarged; a biopsy confirmed the diagnosis. The heart and kidneys were apparently normal. The clinical and laboratory findings were typical. The child was vagotonic. There was glycogen storage in the leukocytes of the blood, but not in the leukocytes of the bone marrow. X-ray examinations revealed multiple dark lines in the metaphyses of the long bones and also some small round areas of decalcification in the cranial bones, due perhaps to displacement of osseous tissue by glycogen deposits.

HIGGINS, Boston.

SOME THEORIES ON TREATMENT OF ACUTE DIARRHEA IN INFANTS. J. A. DE ARGUMOSA, Acta pediat. españ. **9**:45 (Jan.) 1951.

This is a review of Spanish, German, and [one] British investigators in the field of the diarrheas in infancy.

The various theories as to cause and treament are classified as (1) vagotonic, with treatment directed at the vagus nerve and the sympathetic nervous system; (2) diarrhea as an intestinal lesion, with therapy of bismuth salts and tannic acid derivatives, and (3) diarrhea as a nutritional disease, with therapy consisting of high-protein diet, pectin and high-vitamin intake—first with "concentrates," and later in foods. Sulfonamides and antibiotics are used for any infections occurring concomitantly. In the third classification the author discusses certain protein milks and other proprietary foods used in Spain.

Dehydration and the loss of electrolytes necessary to life and health are given as a fourth cause of diarrhea. The rationale of various solutions and their chemical reactions are discussed. Plasma is advised for severe dehydration because it supplies not only the electrolytes but also the proteins which would prevent hypoproteinemia, likely to follow injudicious use of isotonic sodium chloride solution and other artificial solutions. Plasma substitutes are also discussed.

The last explanation for a continued diarrhea is Newman's theory of the toxic effect of histamine on the gastrointestinal tract. The author feels that the proper use of other therapeutic measures should prevent excessive histamine formations. The treatment if one accepts this theory is the addition of antihistamines to other therapeutic agencies.

ALLEN, Santa Fe, N. M.

RELATIONSHIP BETWEEN PROLONGED QT INTERVAL AND HYPOCHLOREMIA IN PYLOROSPASM. KURT WEISS, Monatsschr. Kinderh. **99**:369 (Oct.) 1951.

Prolonged Q-T intervals, abnormal S-T segments and T waves were noted in infants with pylorospasm when blood-chloride levels were below 350 mg. per 100 cc. and were not seen when above this level. This was also found in infants with other diseases accompanied by vomiting and diarrhea. Without blood-chloride changes the electrocardiogram was not influenced in such manner by blood-alkali reserve, blood potassium levels, and dehydration.

HARTENSTEIN, Medical Corps, United States Army.

CYSTIC FIBROSIS OF THE PANCREAS. O. THALHAMMER, Wien. klin. Wchnschr. **62**:438 (June 15) 1951.

A boy aged 8 years 6 months showed participation of three organ systems as intestines, lungs, and liver, partly simultaneously, partly at different times. He was first seen at the age of 18 months with a persistent cough following measles, and a celiac disease was suspected. The history is given in detail, and, since it is the first reported case from Austria, a discussion of various aspects of the disease is given. The name dysporia entero-broncho-pancreatica congenita familiaris given by Glanzmann is preferred.

AMBERG, Rochester, Minn.

DIARRHEA OBSERVED IN INFANTS IN CONNECTION WITH PSEUDOMONAS AERUGINOSA. BELSKA, MITERA and PATERA, Pediat. listy **4**:244, 1949.

At the end of the year 1948 there was an epidemic of diarrhea at the first children's clinic, which occurred especially in premature and atrophic children. In all the 24 cases Pseudomonas aeruginosa was cultivated from the stools. The same bacillus was also found in 13 out of 15 examined bottles with remnants of milk and was probably the cause of the diarrhea. In serious cases streptomycin was given with good results. Two children died. Ps. aeruginosa can also be cultivated from the stools of healthy persons, but it can, nevertheless, not be considered harmless. In suitable conditions, especially in premature and atrophic children, it may cause a serious illness.

FROM THE AUTHORS' SUMMARY.

OUTCOME AND TREATMENT OF CELIAC DISEASE. LUDMILA HOSTOMSKA and BEDRICH HORAK, Pediat. listy **4**:253, 1949.

The authors followed up through clinical, laboratory, and x-ray methods the outcome of 16 cases of celiac disease, for which treatment had been given in the years 1934 to 1944 in the second pediatric clinic of Prof. J. Brdlik in Prague; these cases were investigated at least four years after the last treatment. Seven children were cured after three to five years; in three cases the latent disease persists even after eight years. In half of the cases the authors found a weight and height deficit, an irritable and stubborn nature and an inclination to upper respiratory infections. In all cases the foot arches were lowered, the abdominal wall was weak, and the teeth were bad; in the majority of cases the chest was flat and Harrison's groove was present. The importance of preventing these sequelae is stressed. The authors feel that particularly in the more serious cases the most important factor in treatment is diet, which, through lack of other possibilities, must relatively soon cover the necessary caloric intake through starch. They recommend careful addition of new types of food and adhering to a diet for a long time. They believe that large doses of "Becozyme" (1 cc. contains 1 mg. thiamine hydrochloride, 2 mg. riboflavin, 20 mg. nicotinamide, 2 mg. pyridoxine and 3 mg. pantothenic acid) and liver extracts improve the results in the treatment of celiac disease.

FROM THE AUTHORS' SUMMARY.

EPIDEMIC JAUNDICE IN CHILDHOOD. J. PROCHAZKA, A. TRAPLOVA, J. HOMOLKA, and K. GROSS, Pediat. listy **6**:142, 1951.

A summary of new clinical experiences with epidemic jaundice is given. Attention is drawn to new epidemiologic measures. A survey of experiences drawn from 1,524 cases is given with regard to practical needs of the pediatrician.

GRULEE, Evanston, Ill.

DUODENAL ATRESIA AND STENOSIS. T. EHRENPREIS and P. SANDBLOM, Acta pædiat. **38**:109, 1949.

Twenty cases of intrinsic duodenal obstruction in the newborn operated upon at Kronprinsessan Lovisa Children's Hospital since 1936 are reported. A brief survey of the pertinent literature is given. Clinical and roentgenological findings, treatment, and results of treatment are discussed on the basis of the series presented. The importance of preoperative and postoperative treatment and especially of maintenance of the fluid and nutritional balance is stressed. Postoperative feeding through an indwelling transanastomotic jejunal tube has proved a simple and efficient means of accomplishing this.

FROM THE AUTHORS' SUMMARY.

HYPERTROPHIC PYLORIC STENOSIS—A SURVEY OF ONE HUNDRED THIRTY-SIX SUCCESSIVE CASES—WITH SPECIAL REFERENCE TO TREATMENT WITH "SKOPYL" (METHYLSCOPOLAMINE NITRATE). NILS MALMBERG, Acta pædiat. **38**:472, 1949.

A survey is made of 136 cases of pyloric stenosis observed in the years 1934 to 1948. In 22 cases (16%) surgical treatment was undertaken with no deaths. Mortality for the entire series was 1.5% (2 cases).

With regard to the pathogenesis of the disease, the author suggests that it is to be sought in some prenatal maternal influence on the child (hormonal?) which leads to the rapid postnatal development of muscular hypertrophy in the distal section of the canalis region of the stomach. This hypertrophy leads in its turn to secondary mucous membrane changes which bring about the transition of the disease from the latent to the manifest stage.

The author reports on the results of conservative therapy with "skopyl" solution (methylscopolamine nitrate). This treatment has greatly reduced the need for surgical intervention in this disease.

FROM THE AUTHOR'S SUMMARY.

STAUB-TRAUGOTT EFFECT IN HEALTHY CHILDREN AND IN CHILDREN WITH CELIAC DISEASE. OLLI SOMERSALO, Acta pædiat. **38**:579, 1949.

Twenty-one double intravenous glucose tolerance tests were performed on healthy children below 7 years of age. The dose varied from 0.5 to 2 gm. per kilogram of weight, and the interval between the injections from 40 to 200 minutes. The first and second curves of the double tolerance tests were usually identical—the Staub-Traugott effect was not obtained. In the 20 double tolerance tests performed by the peroral route this effect was clearly obtained in 10 experiments. In four experiments the first and the second curve reached approximately the same level, and in six there was clearly an inverted Staub-Traugott effect. Eight celiac disease patients on whom intravenous tests were performed reacted to the Staub-Traugott effect in the same way as the healthy children.

FROM THE AUTHOR'S SUMMARY.

EPIDEMIC INFANTILE DIARRHEA AND VOMITING. GUNNAR LAURELL, J. HENNING MAGNUSSON, ERIK FRISELL, and BIRGITTA WERNER, Acta paediat. **40**:302 (July) 1951.

During the autumn of 1949 and the winter and spring of 1950, 62 children with epidemic gastroenteritis were treated at the Sachs Hospital for Children. In connection with the disease a special coli strain, termed B.C.N., was isolated. Forty-one of the children became ill in connection with two outbreaks that occurred in the hospital during the autumn of 1949. The control series consisted of children who were under treatment in the hospital at the same time but did not have diarrhea or vomiting attacks. This control group consisted of 346 children. Among these, three healthy carriers were detected. As regards the clinical picture, the dominating symptoms were loss of weight, anorexia, toxemia, diarrhea and vomiting, severe dehydration, and the risk of a recurrence. Penicillin, sulfonamide compounds and streptomycin were all tried, but failed to produce any specific therapeutic effect. As it was found that the B.C.N. strains were sensitive to aureomycin, treatment with this antibiotic was instituted. Thirty-two children have been treated up to the present. The effect was satisfactory even in very severe cases, and no deaths have occurred.

The bacteriological investigations proved that even after a few days' treatment the B.C.N. strains were no longer demonstrable. The micro-organisms most commonly occurring in connection with the aureomycin treatment were Candida albicans, Pseudomonas aeruginosa and Proteus vulgaris. In most of the cases the flora rapidly returned to normal after the treatment was terminated. So far as the aspect of the spread of the disease is concerned the most interesting observation made was that B.C.N. was very often demonstrable in the respiratory tract. Special note should be made of the fact that it was relatively often found in the nose; this is particularly noteworthy in view of recent investigations into the question of dangerous carriers of disease, in which it was shown that those with bacteria in the nose must easily spread the infection to their surroundings. It therefore seems very probable that the spread of B.C.N. may take place in the form of an air-borne infection. Analysis of the air proved, also, that on two occasions at

least, in connection with the outbreaks in the hospital, B.C.N. was present in the air. Examination of other factors in the environment, such as milk, rubber teats, and thermometers, gave negative results.

The serological investigation for the purpose of demonstrating agglutination was not begun until after treatment with aureomycin had been started and the clinical course of the disease thereby considerably curtailed. Agglutination was demonstrated in the serum in roughly 45% of the children. In the majority of cases, however, it was present in dilutions as low as 1/40 to 1/80. Earlier investigations have shown that antibodies do not pass from mother to infant, and the infant therefore completely lacks antibodies against coli bacteria at birth. The occurrence of agglutination suggests, therefore, that antibodies were acquired after birth, and as adult pregnant women were not found to have antibodies against B.C.N. the infants themselves must have produced them.

The part played by B.C.N. in epidemic gastroenteritis is still arousing discussion. Observations made in the present investigations would seem to indicate that B.C.N. plays a leading part in the course of the disease. It was noted that the rapid clinical improvement and the disappearance of the bacteria from the stools ran parallel with one another. Infections occurred in the hospital at the same time as the bacteria were detected in the ear. On no occasion did B.C.N. carriers who received treatment and were freed from the bacteria give rise to secondary cases. Finally, the serological examinations showed that healthy newborn infants do not possess antibodies against coliform bacteria, and to a certain extent, also, it was possible to demonstrate agglutinins against B.C.N. in infants that had been affected.

The diagnosing of infection with B.C.N. proved to be of great practical significance. By establishing the presence of these bacteria it was possible to select cases suitable for aureomycin therapy and to follow the result of the treatment. With the aid of regular control examinations it was also possible to prevent the disease from breaking out again in the hospital. It could also be ascertained when the infants were free from infection and could be returned without risk to various institutions. All these advantages are possible, however, only at hospitals possessing an effective isolation department where newly admitted children can be examined and come under treatment.

FROM THE AUTHORS' SUMMARY.

FIBROCYSTIC DISEASE OF THE PANCREAS: A DISORDER OF THE AUTONOMIC NERVOUS SYSTEM. HILDING BERGSTRAND, Acta paediat. **40**:350 (July) 1951.

The author describes two cases of cystic fibrosis of the pancreas. The first case, that of a girl 7 weeks of age at death, showed agenesis of the ganglion cells in the distal ileum and in the entire colon and rectum. No Paneth cells were present in the small intestine. In the second case, that of a girl 20 months of age at death, no abnormalities of the sympathetic nervous system could be found on microscopic examination. As in some previously described cases, the mucous glands of the respiratory tract in the second case were distended and contained inspissated secretion, but goblet cells were practically absent. Consequently, the microscopic appearance of the lungs was quite dissimilar to that in asthma, a disease in which the parasympathetic nervous system is believed to be overstimulated. The author draws the conclusion that both of his cases strongly support Farber's theory that the basic disorder of cystic fibrosis of the pancreas is an imbalance of the autonomic nervous system. He believes, however, that the cases do not tally with Farber's conception of the disorder as hyperfunction of the parasympathetic nervous system but that they suggest it to be a dysfunction or a hypofunction of this system.

FROM THE AUTHOR'S SUMMARY.

PRIMARY HEMANGIOENDOTHELIOSARCOMA IN THE LIVER OF CHILDREN. O. STEINICKE NIELSEN, Acta paediat. **40**:431 (Sept.) 1951.

A brief review is given of the etiology and of previously reported cases of primary endotheliosarcomas of the liver. A case is described of hemangioendotheliosarcoma in a boy almost 12 months old, who died after three weeks of illness. An autopsy was performed. A brief account of the course of illness is given.

FROM THE AUTHOR'S SUMMARY.

ONE HUNDRED CASES OF PYLORIC STENOSIS. T. J. HALBERTSMA, Maandschr. kindergeneesk. **17**:213, 1949.

The author reviews 100 definite cases of surgically treated hypertrophic pyloric stenosis seen among 7,000 clinical cases; in 30 other cases operation was not necessary. In the series there were 89 boys and 11 girls. The youngest child was 2 weeks, the eldest 16 weeks old at the time of operation.

During the war very few cases occurred; afterwards strikingly many. There was a strong familial tendency; five times there were 2 cases in one family, once even 3 cases. Two of these families were, moreover, related, the 4 children being first cousins.

All the children but one were cured; this was a 10-week-old baby, who entered the hospital in a very bad condition.

The pathogenesis of pyloric stenosis is still unknown. Because in the preoperative period many patients were cured after conservative treatment, the author believes that the Ramstedt operation is only a kind of "first aid" by which the time between the spastic period and the following natural recovery is bridged over.

FROM THE AUTHOR'S SUMMARY.

CONGENITAL OCCLUSION OF THE DUODENUM AND JEJUNUM. P. P. M. NIEUWENHUIS, Maandschr. kindergeneesk. **17**:223, 1949.

The clinical and roentgenological appearance of congenital occlusion of the duodenum and the upper part of the jejunum are described. In four cases the continuity of the intestine was established or restored, with two recoveries. An early diagnosis is of primary importance to get good results.

FROM THE AUTHOR'S SUMMARY.

Nervous Diseases

NERVOUS ENCEPHALOCELES: SURVEY OF PROBLEMS, WITH RECOMMENDATIONS FOR REDUCING MORTALITY. E. WALKER, W. W. MOORE, and J. R. SIMPSON, A. M. A. Arch. Otolaryng. **55**:182 (Feb.) 1952.

Intranasal encephalomeningocele probably occurs much more frequently than is indicated by the number of reported cases. A review of the literature indicates that the occurrence of intranasal encephalocele has been associated with an extraordinarily high mortality. The principal causes of this high rate in the past have been failure to appreciate the nature of the lesion and ill-advised intranasal procedures, which have resulted in meningitis.

The first essential in reducing the mortality is a correct diagnosis. Any nasal tumor discovered in a newborn baby or a child is likely to be an encephalocele, since polyps and neoplasms are rare in children. The pedicle always leads to the superior limit of the nasal sulcus. The mass itself often protrudes from one nostril and may be visible in the pharynx. It is soft and compressible and may weep cerebral fluid. Though it may be cystic, aspiration is hazardous because of the risk of meningitis. Rhinorrhea is frequently evident, and the root of the nose may be distended by the intranasal mass.

The authors advise against any intranasal procedure which may initiate leakage of cerebrospinal fluid, once the diagnosis has been made. Adequate chemotherapy should be instituted. If any significant nasal obstruction exists, a tracheotomy is indicated. Intracranial repair of the cerebrospinal-fluid communication should be performed as the primary operation. If there is any persisting nasal obstruction, it can be relieved by the intranasal removal of the hernial mass.

In their view of the literature, Walker and his colleagues found a total of three cases in which intracranial repair of an intranasal encephalocele was successfully carried out. They present their case of the lesion in a baby girl which was similarly treated, with a successful outcome.

ALPERS, Philadelphia [A. M. A. ARCH. NEUROL. & PSYCHIAT.].

NATURE AND RECOGNITION OF CEREBRAL PALSY IN INFANCY. MEYER A. PERLSTEIN, and HARRY E. BARNETT, J. A. M. A. **148**:1389 (April 19) 1952.

Pathologic conditions in the motor cortex and motor pathways within the brain manifesting pyramidal signs and called cerebral palsy are discussed from the point of view of etiology, various

manifestations, history, and symptoms. Methods for examining the motor system of small infants are described in detail. Fifty per cent of those who were mostly spastic and 25% of those who were mostly athetoid had associated mental retardation.

KIMBALL, Evanston, Ill.

INFANTILE CEREBRAL PALSY. M. A. PERLSTEIN, J. A. M. A. **149**:30 (May 3) 1952.

An attempt is made to define cerebral palsy. Then a method of classification is logically presented and described. It is recommended that attempts be made to classify all cases according to (1) anatomic site of brain lesion, (2) clinical symptoms, (3) topographical involvement of extremities, (4) degree of muscle tone, (5) severity, and (6) etiology. Cognizance is taken of the difficulties with (1) because of the changing knowledge concerning physiologic-anatomic sites in the brain. Little consideration is given to the disturbing effects of growth upon items 2, 3, 4, and 5.

KIMBALL, Evanston, Ill.

EVALUATION OF VITAMIN E THERAPY IN DISEASES OF THE NERVOUS SYSTEM. SIMON STONE, J. Nerv. & Ment. Dis. **3**:139 (Feb.) 1950.

Vitamin E as present in wheat germ oil and natural mixed tocopherols contains myotrophic and neurotrophic factors promoting capillary permeability. It has been found useful in treatment in some cases of muscular hypotonia and hypoplasia in the young, in anterior poliomyelitis, and in polyneuritis. It serves to arrest or slow down the progress of demyelinization when used in combination with B complex in diseases like amyotrophic lateral sclerosis and multiple sclerosis.

In some cases of congenital nonobstructive hydrocephalus wheat germ oil produces arrest or slowing down of progression of head enlargement with subsequent normal development. Its use is suggested as a necessary vitamin supplement for children who show a defective maturation of their neuromuscular apparatus.

JAHR, Omaha.

PRECOCIOUS PUBERTY AND TUMOR OF THE HYPOTHALAMUS. C. P. J. STOTIGN and W. J. H. NAUTA, J. Nerv. & Ment. Dis. **3**:207 (March) 1950.

A case is reported of precocious puberty, and the subject is discussed from the viewpoint of psychosomatic changes resulting from tumor pressure and destruction of tissue in the hypothalamic area. The boy was followed from the age of 6 years, 11 months, to the age of 10 years, 8 months. The symptoms began during the fourth year of life, when an abnormally rapid growth became manifest, with marked development of the genitalia. During the years of observation the main features were marked retardation of the previously accelerated body growth, absence of major neurologic symptoms, deteriorating epilepsy, arrest of mental development, development of dementia, and behavioral aberrations.

JAHR, Omaha.

EARLY FAMILIAL CEREBELLAR DEGENERATION: REPORT OF THREE CASES IN ONE FAMILY. GEORGE A. JERVIS, J. Nerv. & Ment. Dis. **3**:398 (May) 1950.

All three siblings, varying in age from 2 years to 9 years, were mentally deficient with signs of cerebellar dysfunction. The youngest of the set died at the age of 2 years. Autopsy showed extensive degeneration of the granular layer and to a lesser degree of Purkinje's cells. There was involvement of the inferior olive. The central white matter of the cerebellum, the dentate nuclei, the pons, and the red nuclei were normal.

The children were Italian, and there was no significant or even suggestive family history on either side of the parental tree.

JAHR, Omaha.

FAMILIAL HAEMANGIOMATA OF THE CEREBELLUM. H. O. TONNING, R. F. WARREN, and H. J. BARRIE, J. Neurosurg. **9**:124 (March) 1952.

Hemangioblastomata of the central nervous system are isolated lesions unless they constitute part of the hereditary disease known as the von Hippel-Lindau syndrome, when they may be associated with angiomata of the retina and spinal cord, multiple cysts of the kidneys and pancreas, and even renal carcinoma.

The authors stress the importance of taking a careful family history when vascular tumors are discovered in the brain. Three cases of the Lindau-von Hippel syndrome among siblings in a family of four children are reported.

One patient died after removal of a cerebellar tumor and was found to have cysts in the lung, kidney, and pancreas. One died of renal carcinoma after successful removal of a cerebellar angioma. One has had a successful removal of a cerebellar angioma.

The authors believe that the condition must be due to a hereditary, but persistent, abnormality in which there is a lack of integration between blood vessels and their field of supply. In the phase of fetal development this leads to cysts, and in later life to neoplasms.

ALPERS, Philadelphia [A. M. A. ARCH. NEUROL. & PSYCHIAT.].

REVIEW OF SURGICAL TREATMENT OF INTERNAL HYDROCEPHALUS. IRA J. JACKSON, J. Pediat. **8**:251 (Feb.) 1951.

For noncommunicating hydrocephalus, the ventriculocisternostomy of Torkildsen and the third ventriculostomy of Dandy are at present the most popular methods of treatment. For communicating hydrocephalus, ingenious new methods for shunting excess cerebrospinal fluid have been devised. It is too soon to say which type of shunt will be most successful. In this type of hydrocephalus, coagulation of the choroid plexus has been most universally used. It has been successful in about 50% of selected cases.

MACDONALD, Pittsburgh.

SUBDURAL HEMATOMA, HYGROMA, AND HYDROMA IN INFANTS. GORDON KINLEY, HARRIS D. RILEY JR., and CLAUDE S. BECK, J. Pediat. **8**:667 (June) 1951.

Birth trauma is probably the commonest contributing factor to abnormal collections of subdural fluid. Subdural hematoma is a collection of bloody and/or xanthochromic fluid encapsulated by a definite neomembrane. Hygroma is a collection of xanthochromic fluid free in the subdural space with a nonencapsulating membrane. Hydroma is a collection of colorless or slightly xanthochromic fluid free in the subdural space with no evidence of membrane. All three probably represent stages in healing of subdural or intradural hemorrhage. Of the three, hydroma is the least commonly encountered.

The authors have reviewed the literature and report on 22 additional patients. The signs and symptoms are not particularly diagnostic. As has been pointed out by others, irritability, convulsions, vague gastric intestinal symptoms, and failure to gain weight are frequently encountered. Of course, the demonstration of abnormal collections of fluid in the subdural space, either by subdural puncture alone or by pneumoencephalography, is the positive diagnostic test.

Once the diagnosis is established, repeated taps may be enough to improve the condition of the patient. Many of these collections of fluid are bilateral, and a fair number, for which treatment is by tap alone, recur. The authors advise routine bilateral osteoplastic flaps in all cases. The capsule of subdural hematoma should be removed. Since it is almost impossible to remove the membrane of hygroma and since hydroma has no membranes, these two conditions are met by stripping the inner layer of the dura.

The authors stress the advisability of evaluating concurrent intrinsic brain damage before operation.

MACDONALD, Pittsburgh.

ABDOMINAL PAIN AS A MANIFESTATION OF EPILEPSY. SAMUEL LIVINGSTON, J. Pediat. **8**:687 (June) 1951.

The syndrome of "abdominal epilepsy" is relatively new, although the association of abdominal pain with known epilepsy has been recognized for years. Livingston here reports 14 children with recurrent paroxysmal attacks of abdominal pain considered to be cerebral in origin. All of these children presented one or more of the following features: recurrent attacks of abdominal pain followed by exhaustion and sleep; electroencephalographic abnormalities, and/or a favorable clinical response to diphenylhydantoin sodium.

MACDONALD, Pittsburgh.

BRAIN TUMORS IN CHILDREN. ARTHUR B. SMITH, Radiology **58**:688 (May) 1952.

Smith reviews the literature concerning brain tumors in children and adds 34 cases of proved cerebral tumors. Approximately one-sixth of all tumors of the brain occur in children under 15 years of age. Most of these occur in the younger age groups and are located chiefly below the tentorium cerebelli. The majority of the subtentorial tumors are highly malignant. The commonest subtentorial tumor in children is the astrocytoma, which is benign and grows slowly, usually in one cerebellar hemisphere. If removal can be accomplished, it does not recur.

The medulloblastoma is the second in order of frequency. Medulloblastomas are commonest in boys from 3 to 6 years of age. Irradiation is the treatment of choice for cerebellar medulloblastoma, although it is not curative. Operative treatment rarely gives relief for more than six months, no matter how radical the operation. The striking improvement which is usually noted within 10 days of the beginning of x-ray therapy is of some diagnostic value, for other tumors in this region are not nearly so radiosensitive.

Ependymoma of the fourth ventricle is about one-third as common as medulloblastoma. Tumors of the cerebral hemispheres are uncommon in children. In the author's series, 11 tumors were supratentorial, and 23 were subtentorial. The supratentorial tumors usually occur in or around the third ventricle. Tumors around the third ventricle all produce increased intracranial pressure, and treatment is generally unsatisfactory. Tumors of the pons and medulla are usually highly malignant, infiltrating gliomas, which are resistant to all forms of treatment.

Plain films of the skull reveal abnormality in more than half of the cases of brain tumor in children, whether by evidence of increased intracranial pressure, abnormal calcification, or bone erosion. Ventriculography was performed in 16 of the author's cases. In 12 of these 16 cases the diagnosis of a localized mass was made from the x-ray films. Dilated ventricles were found in the other four cases.

WEILAND, Grove City, Pa. [A. M. A. ARCH. NEUROL. & PSYCHIAT.].

NOTES ON CEREBELLAR ATAXIA IN CHILDHOOD. ROBERT A. SHANKS, Arch. Dis. Childhood **25**:391 (Dec.) 1950.

Thirty-nine cases of cerebellar ataxia over a period of 20 years are analyzed. Thirty of these were traced and the outcome described. The mode of onset of the ataxia was a most valuable prognostic indication. Cases in which onset was sudden had an almost uniformly benign course, while those in which the ataxia began insidiously were mainly of the progressive type; there is little to distinguish the acute, benign form of the disease from the hopelessly progressive one except the onset.

KELLY, Milwaukee.

SYNCOPAL ATTACKS IN INFANCY. WILFRED SHELDON, Great Ormond St. J. (No. 3) 20 (June) 1952.

Repeated attacks of syncope occurring in infants are described, four cases being cited. No investigation as to cause was made, no treatment was given, and no conclusions were arrived at.

McGUIGAN, Evanston, Ill.

HEREDITARY SENSORY RADICULAR NEUROPATHY. D. DENNY-BROWN, J. Neurol., Neurosurg. & Psychiat. **14**:237 (Nov.) 1951.

In 1922 Hicks described a family of 34 members spread over four generations 10 of whom suffered from perforating ulcers of the feet, shooting pains about the body, and deafness. Denny-Brown saw a member of the third generation of this family at the age of 54, during her last illness, and he reports the clinical and necropsy findings.

The postmortem examination disclosed primary degeneration of dorsal root ganglia. Deposits of amyloid were found in the most severely affected ganglia, but they were considered a secondary feature.

In this pedigree the disorder was inherited as a simple Mendelian dominant. The greater loss of sensation of pain and temperature is a result of the radicular pattern of sensory loss. The disease has a sporadic and familial form as well as the hereditary form, and in such cases deafness is absent from the syndrome.

ALPERS, Philadelphia, [A. M. A. ARCH. NEUROL. & PSYCHIAT.].

REVIEW OF STUDIES OF HUMAN FETAL CELLS IN TISSUE CULTURES. MARY JANE HOGUE, Études néo-natales **1**:3 (May) 1952.

Nerve cells do not all die when the individual dies, but some may be kept alive for nine days in the brain before they are cultured, provided that they are kept at a low temperature, 4 to 6 C.

Tissue cultures of fetal brain neurons have been kept alive for 323 days in an artificial culture medium, when they were fixed and stained for study. The neurons migrate out from the explant and move about in the culture medium. Some neurons are known to have a definite polarity. Purkinje cells migrate with the dendritic processes forward and the axis cylinder trailing behind.

Several different types of neurons have been identified in tissue cultures. Many neurons are binucleate, a few are multinucleate, but mitosis and cell division have not been observed in nerve cells of the brain. Microglia, macrophages, oligodendroglia, and two types of astrocytes have been observed and studied. One young fetus contained cells without processes which were interpreted as neuroblasts.

Choroid plexus cells were grown in cultures. The granules were studied with the phase microscope. The formation and pinching off of small clear globules into the culture media were observed. These dissolved and became a part of the fluid medium and were interpreted as apocrine secretion.

Active ciliated columnar ependymal cells were found in vesicles appearing from explants of cerebellar material. They are interesting phylogenetically, as they are found in some lower vertebrates but not in the adult human brain. FROM THE AUTHOR'S SUMMARY.

ANENCEPHALY: PRIMARY OR SECONDARY CONDITION? ANDRE-THOMAS, Presse méd. **58**:1425 (Dec. 25) 1950.

The author describes in exhaustive detail the neurological examination and the autopsy findings in the case of an anencephalic newborn infant. There was complete absence of the pons and of all the cephalad structures of the central nervous system, but in the tissue structures which replaced normal brain tissue there was excessive vascularity, with a large amount of collagen deposition.

Was the destruction of a normally developing nervous system brought about by the overgrowth of this extraneous tissue, or did the blood-vessel invasion and collagen deposit occur as an effort to fill in an empty space?

From the histopathological evidence, Andre-Thomas concludes that some growth at least had occurred in the primative nerve stem.

The illustrations accompanying the article are excellent in their detail.

DAFFINEE, Melrose, Mass.

PURULENT MENINGITIS OF OTO-SINAL ORIGIN IN CHILDREN. ENRIQUE DELUCCHI, Rev. otorrinolaring. **12**:54 (Sept.) 1952.

Delucchi presents a series of 36 patients with purulent meningitis. Of these, 18 patients showed foci in the ear or the sinuses, while the origin was obscure in nine patients. Fifteen of his patients recovered. He stresses the fact that in children, in the absence of a typical epidemic, the commonest etiologic factor for purulent meningitis is in either the ear or the sinuses. Secondly, frequently the focus in this area is extremely latent and difficult to determine and evaluate, and secretions may be sterile. He stresses most careful and rigid examinations by x-ray, sinus puncture, and surgical exploration and most careful otorhinologic studies, and, finally, when the pathological nature is established in either the sinuses or the mastoid, surgical intervention and the use of antibiotics are indicated and should be instituted early.

PERSKY, Philadelphia [A. M. A. Arch. Otolaryng.].

RECENT OBSERVATIONS OF A FORM OF ENCEPHALITIS CLINICALLY AND ANATOMICALLY DISTINGUISHABLE BUT OF UNKNOWN ETIOLOGY. H. PETTE and H. KALM, Deutsche med. Wchnschr. **75**:1173 (Sept. 8) 1950.

Results of histological examination of the brain in three cases are described. Two of the brains came from children 6 and 8 years of age. Focal and nodular increase of glial tissue

and, depending on the acuteness of the lesion, pronounced congregations of lymph and plasma cells around the vessels and mainly around the veins were found. Certain ganglion cell areas are preferred. The picture is complicated by general and local degenerative processes due to circulatory disturbances. Extrapyramidal symptoms predominate. Clinically hyperkinesias of the most varied type occur side by side, such as choreiform and athetoid motions, myoclonus, and torsion dystonias. These may also vary in intensity. Laughing and crying spasms may occur. In all symptoms a certain rhythmicity may be observed. The pyramidal system may also participate. All patients presented some psychic symptoms.

AMBERG, Rochester, Minn.

CLINICAL AND ENCEPHALOGRAPHIC FINDINGS IN CASES OF SUSPECTED VERGA VENTRICLE. H. SCHÖNENBERG, Ztschr. Kinderh. **68**:512, 1950.

The air-encephalogram of malformations of the septum pellucidum are demonstrated. This malformation may be hereditary or may be due to toxic or infectious damage during the prenatal period. No correlation between clinical symptoms and this malformation could be found. The examinations were made on children showing various symptomatic complexes pointing toward evidence of cerebral damage.

GLASER, Jerusalem.

Psychology and Psychiatry

EXPERIMENTAL GLUTAMIC ACID TREATMENT IN MENTALLY RETARDED CHILDREN. VERA OLDFELT, J. Pediat. **40**:316 (March) 1952.

Sixteen patients, aged 7 to 15 years, all with low intelligence quotients, were given 18 gm. of glutamic acid per day in three doses; treatment was continued for a little more than four months. A control group numbered 15 patients. The treatment had no demonstrable effect on the intelligence of these children.

MACDONALD, Pittsburgh.

WHAT IS CHILD GUIDANCE? M. B. GAMAT, Indian J. Child Health **1**:449 (Sept.) 1952.

The author sums up the aims of child guidance as an effort to socialize the neurotic and the aggressive, encourage the dull and retarded, and redeem the delinquent. It is hoped thus to build up integrated personalities with normal adjustment to community life.

MCGUIGAN, Evanston, Ill.

ROLE OF PEDIATRICIAN IN PSYCHOLOGICAL HANDLING OF DIFFICULT CHILD. D. ORIGLIA, Minerva pediat. **4**:80 (Feb. 15) 1952.

A detailed discussion is presented of the familial and environmental factors, the symptoms, the diagnosis, and the method of dealing with the maladjusted child.

HIGGINS, Boston.

Diseases of Genitourinary Tract

THE NEPHROTIC SYNDROME IN CHILDREN: I. CLINICAL RESPONSE TO NITROGEN MUSTARD THERAPY. VINCENT C. KELLY and THEODORE C. PANOS, J. Pediat. **41**:505 (Nov.) 1952.

Nitrogen mustard treatment of nephrosis is based on the speculation that the pathogenesis of the nephrotic syndrome is related to antigen-antibody reactions or to the Shwartzman phenomenon. Kelly and Panos gave nitrogen mustard (HN_2) intravenously in 5% dextrose in water. A course of therapy consisted of a dose of 0.2 mg. per kilogram of body weight on each of two successive days. Diuresis may occur one to eight days after treatment; it appeared here in 69% of cases. There was little effect on serum electrolytes. Unfortunately, edema frequently recurred.

MACDONALD, Pittsburgh.

EFFECTS OF MAGNESIUM SULFATE ON RENAL FUNCTION IN CHILDREN WITH ACUTE GLOMERULONEPHRITIS. JAMES N. ETTELDORF and A. H. TUTTLE, J. Pediat. **41**:524 (Nov.) 1952.

In acute glomerulonephritis, magnesium sulfate produces generalized arteriolar relaxation. This increases effective renal plasma flow and also reduces systemic blood pressure.

MACDONALD, Pittsburgh.

LONG TERM RESULTS OF TRANSPLANTATIONS OF THE URETERS FOR ECTOPIA VESICAE. D. INNES WILLIAMS and H. R. JOLLY, Great Ormond St. J. (No. 3) 9 (June) 1952.

The author studied the results in 45 cases of transplantation of the ureters following operation. The series covered a period of thirty years.

Some impairment of renal function was found in every case postoperatively, and cases coming to autopsy showed advanced disease in one or both kidneys. Unless great care is taken at operations there is great danger of ureterocolic obstruction of varying degrees.

Infection of kidney urine is probably present in the majority of cases, though severe pyelonephritis occurs only occasionally. Of 30 traced patients surviving more than two years, 9 had periodic attacks of fever with pain in the loins. All the deaths were attributed to renal disease, inflammation rather than obstructive changes being the important factor.

Blood urea apparently becomes stabilized most often between 40 and 70 mg. per 100 cc. In only 7 of 16 was it within normal limits.

Incontinence is frequently fairly well controlled during the day but not often well controlled at night. One patient surviving 21 years has no more than two dry nights a month. Of 30 patients 9 had nocturnal lapses.

Postoperative survival is strikingly shown in the final table. Eighteen of 22 patients were still alive after five years. After 10 years 10 of 13 traced patients survived; after 15 years, 7 out of 11, and after 20 years, 2 out of 5.

McGUIGAN, Evanston, Ill.

DIFFUSE BILATERAL RENAL LYMPHOSARCOMATOSIS. J. G. MILLICHAP, Great Ormond St. J. (No. 3) 76 (June) 1952.

Two cases of lymphosarcomatosis with diffuse bilateral renal involvement are reported.

McGUIGAN, Evanston, Ill.

CASE OF PUBERTAS PRAECOX. P. BERGMAN, Nord. med. **39**:1407 (July 23) 1948.

A girl of 5 years showed signs of puberty from her sixth month of age. The menstruation cycle was normal after the thirteenth month of age. Oophorectomy was performed without any influence on the puberty development. In the ovary a corpus luteum cyst was found. The etiology of the premature development of puberty was unknown. Probably the case belongs to the so-called constitutional types of pubertas praecox.

WALLGREN, Stockholm.

Diseases of Ductless Glands; Endocrinology

ANTIDIURETIC SUBSTANCE IN THE BLOOD OF NORMAL AND ADRENALECTOMIZED RATS. JAMES H. BIRNIE, W. J. EVERSOLE, W. R. BOSS, C. M. OSBORN, and ROBERT GAUNT, Endocrinology **47**:1 (July) 1950.

In the blood serum of normal and adrenalectomized rats there is a substance which depresses the rate of urine flow and increases chloride excretion.

It exerts its antidiuretic action by causing an increased renal tubular reabsorption of water despite causing a transient rise in the glomerular filtration rate. Its effects are not duplicated by foreign proteins. It is not present in the blood of hypophysectomized rats, and in all respects tested its actions are like those of posterior pituitary extract. Its pituitary origin is suggested. The substance accumulates after adrenalectomy, and this fact plus the demonstrated hypersensitivity to antidiuretic agents helps to explain the absence of normal water diuresis

after adrenalectomy. The accumulation of the antidiuretic substance after adrenalectomy is readily prevented by replacement therapy with saline solution, desoxycorticosterone acetate, or adrenal cortical extract.

JACOBSEN, Buffalo.

FATE OF C^{14}-TESTOSTERONE and C^{14}-PROGESTERONE IN MICE AND RATS. MICHAEL C. BARRY, MAXWELL LEIGHT EIDINOFF, KONRAD DOBRINER, and T. F. GALLAGHER, Endocrinology **50**:587 (June) 1952.

After the intraperitoneal injection of C^{14}-4-testosterone and C^{14}-21 progesterone in mice and rats, the radioactive material was rapidly excreted in both urine and feces. Practically all the administered radioactive material was recovered. It is clear that the male hormone, testosterone, and the female hormone, progesterone, remain such a short time in the rodent that the radiation from the isotope does not constitute any reasonable threat to the animal. Moreover, the hormone and its metabolites were not significantly accumulated by any of the organs or tissue examined, and, therefore, damage to a particular target area would not be expected. The steroid hormones thus are not stored in the gland like the thyroid hormone, and testosterone has a short biological half-life in contrast to a sterol like cholesterol. On the basis of these studies C-labeled testosterone is a relatively innocuous substance in rodents. The conclusion was drawn that the fate of these hormones in rodents does not afford information directly applicable to the problem of steroid hormone metabolism in human beings.

JACOBSEN, Buffalo.

SOME DIFFERENCES BETWEEN TWO STRAINS OF RATS DEVELOPED BY SELECTION TO DIFFER IN THEIR RESPONSE TO EQUINE GONADOTROPHIN. L. E. CASIDA, BETTY R CASIDA, and A. B. CHAPMAN, Endocrinology **51**:148 (Aug.) 1952.

A comparison has been made of two inbred lines of rats developed by generations of selection to differ in the response of their ovaries to a standard dose of equine gonadotropin. The findings suggest that the differences in response of the two lines are due largely to a difference in the amount of ovarian tissue at the time of treatment.

JACOBSEN, Buffalo.

PINEALECTOMY VS. PINEAL INJECTION IN THE YOUNG COCKEREL. C. J. SHELLABARGER, Endocrinology **51**:152 (Aug.) 1952.

The pineal body remains an organ of doubtful endocrine function. Examination of review articles discloses that the following conflicting claims are found in the literature. 1. Pinealectomy has been claimed to cause any one of three effects on the gonads, hypertrophy, or atrophy, or no effect. 2. The same claims have been made following addition of pineal material by implantation or injection, hypertrophy, or atrophy, or no effect on gonad weight.

Experiments on cockerels reported in this paper showed that pinealectomy resulted in inhibition of testis growth, the injection of pineal material into chicks not operated on resulted in an increased testis growth, and the injection of pineal material into pinealectomized chicks restored the testis weights to normal.

JACOBSEN, Buffalo.

CARCINOMA OF THE THYROID IN CHILDREN. RICHARD H. LAWLER, JAMES W. WEST, and EDMUND G. LAWLER, Illinois M. J. **101**:322 (June) 1952.

A report is made of two cases of papillary and solid carcinoma of the thyroid which occurred in children of 4½ and 7½ years of age.

BARBOUR, Peoria, Ill.

PROTECTIVE EFFECT OF DIETARY FAT ON IMMATURE RATS FED THYROID. SAMUEL M. GREENBERG, J. Nutrition **47**:31 (May 10) 1952.

Methyl linoleate and cottonseed oil administration protect the immature rat against growth retardation when the rat is fed a fat-free, sucrose-casein diet to which thyroid has been added. Vitamin B_{12} has no appreciable effect in counteracting the growth-retarding action in rats of desiccated thyroid powder in a fat-free, sucrose-casein diet. The possible relationship between the protective activity of fat and that of liver for immature rats fed thyroid is discussed.

FREDEEN, Kansas City, Mo.

THE LAURENCE-MOON-BIEDL SYNDROME. I. KESSEL, J. Pediât. **40**:96 (Jan.) 1952.

This is the first recorded case of the Laurence-Moon-Biedl syndrome in a pure South African Bantu. The patient is a girl aged 5 years 6 months. With the exception that although the patient had retinitis pigmentosa there was no perivascular pigment as in typical retinitis pigmentosa, with the bone corpuscle appearance, the case was almost typical. MACDONALD, Pittsburgh.

PERSONALITY DEVELOPMENT IN DYSTROPHIA ADIPOSOGENITALIS. GION CONDRAU, Helvet. paediat. acta **4**:415 (Nov.) 1949.

The author reports on the post-treatment control of 31 cases of dystrophia adiposogenitalis. The patients were treated in the Kinderspital Zurich in the years 1925 to 1934 and reexamined in 1947 and 1948.

The course of the physical disease was as follows: In 5 cases obesity was remarked at birth; in 9 cases it appeared during the first six years of life, and in another in the eighth year. In 14 cases the obesity appeared between the ages of 9 to 13. (In 2 cases it was not possible to give a definite date of commencement.) In 17 cases the obesity remained unaffected by hospital treatment and continued after the patient had grown up; in 4 cases a considerable, and in 2 cases a slight, permanent improvement was achieved by the clinical treatment, while in 3 cases there was temporary improvement. In only 3 cases was obesity permanently cured. (Two patients died young.) The genital underdevelopment ceased in almost half the cases by the time that the patient had grown up. There was no obvious connection between the cessation of the genital underdevelopment and that of the obesity.

In general the patients reexamined showed peculiarities of personality as regards energy and temperament. They were generally good-tempered and sluggish, lacked initiative, vitality, and independence, but suffered from irritability, depressions, and other dysphoric moods. These findings are confirmed by earlier accounts, which describe the obese as resigned, uninterested, patient, obedient, good-tempered, but moody, without self-confidence, querulous, and irritable. The so-called "hypophysical temper" on which von Frankl-Hochwart remarked—that is, a carefree contentment, apathy, and resignation—appeared in a number of these patients also. A number of patients with slight intellectual debility were seen, particularly in the forms which commenced in early childhood. The moodiness and sluggishness which are so frequent in dystrophia adiposogenitalis fit into the picture of the psychopathological-diencephalic syndrome. It is true that nontypical psychic disturbances also occur. The physical disturbances seem often to cause neurotic developments which can hardly be distinguished from the direct hormonal or diencephalic influences. A definite correlation between the degree and type of psychic disturbance and the degree of physical disturbance could not be proved in so small a number of patients.

The near relatives of the patients do not appear to suffer from frequent psychoses, though accounts suggest frequent cases of obesity and psychopathic peculiarities of character among them.

FROM THE AUTHOR'S SUMMARY.

ADDISON'S DISEASE. OSCAR CORREA and ATILIO PIERA, Rev. chilena pediat. **23**:110 (March) 1952.

A case of Addison's disease in a girl aged 11 years 6 months is described. The patient was admitted during an acute adrenal crisis, from which she promptly recovered on the usual treatment with cortical extract and Ringer and glucose solutions. The diagnosis was based on the characteristic signs of the acute episode and on hyperpigmentation of skin and gums, low excretion of 17-ketosteroids and 11-oxysteroids, positive Robinson-Power-Kepler and Thorn tests for corticoadrenal insufficiency, and calcification of one of the adrenal glands. After the acute crises had been corrected, desoxycorticosterone, salt and streptomycin were used. The patient remained in good physical and mental health for about three and one-half months; a rather sudden picture of heart failure then developed, and the patient died. At the postmortem examination a total destruction of both adrenal glands was found, the glandular tissue appearing to be replaced by fibrocaseous masses of tuberculous origin.

FROM THE AUTHORS' SUMMARY.

MAURIAC'S SYNDROME IN DIABETIC CHILDREN. MARIA LUISA SALDUN DE RODRIGUEZ, Arch. pediat. Uruguay **20**:587 (Sept.) 1949.

Mauriac's syndrome, which has also been called thesaurismosis in childhood or juvenile diabetes, polycoria of diabetic children, liver enlargement in juvenile diabetes, diabetic dwarfism with enlargement of the liver, and enlargement of the liver with delay in growth in diabetic children, has been observed by the author in nine cases which are reported. It is generally seen in diabetic children whose disease began in infancy, but it also is found in those in whom the disease began at a later age; it is always preceded for some time by diabetes (one to various numbers of years). It is exceptional in adults. Another condition for its appearance is a maladjusted and uncontrolled diet. Its characteristics are a delay in growth, sometimes total; big abdomen, due almost always to a huge enlargement of the liver without great changes in the functioning of this organ, although the diabetic with Mauriac's syndrome has a decrease in the defense of the liver that predisposes him to hepatitis; functional lumbar lordosis; fat infiltration and edema of the hypodermic tissue; painful abdominal crises; hyperglycemia (sometimes rising to enormous values); glycosuria in variable form; hypercholesteremia; delay in the age of ossification and in sexual development, and some degree of mental apathy. The three most characteristic symptoms are the beginning of diabetes at an early age, the delay in growth, and the enlargement of the liver. The cases of Mauriac's syndrome differ from those of von Gierke's disease by the blood-chemistry picture; in the former there are hyperglycemia and glycosuria, and in the latter, hypoglycemia and low curve in the glucose tests. In addition, the test with epinephrine gives different results in the two diseases. However, cases of both diseases have been described in the same family. As to the pathological changes, it was held until a short time ago that the large liver in the diabetic person with thesaurismosis is poor in glycogen while that in von Gierke's disease is rich in this substance. Recent papers are mentioned in which different and opposite points of view are shown and lead to acceptance of a part of each of the opinions expressed. The pathogenesis of this syndrome has been related to the intensity and duration of the treatment with insulin (Mauriac, who considered the syndrome always of bad prognosis); others admit that the treatment acts so as to permit the survival of the diabetic and the longer course of the disease, especially in small children, and consequently the appearance of this syndrome; others consider the syndrome due to the lypocaic hormone. Studies of the author revealed that Mauriac's syndrome appears always in children whose diabetes has begun very early, and appears after more than a year of disease; none of the patients followed a well adjusted and controlled diet, being generally children on free diets with an insufficient quantity of insulin; none had received protamine zinc insulin or other mixtures of retarded action; all had permanent glycosuria and consequent high hyperglycemia before eating; in most cases there was hypercholesteremia. All the patients were put on a sufficiently balanced diet, exactly calculated in values and equivalents. Insulin doses were increased and adjusted to the necessities of the case, insulin of slow action being used (protamine insulin or, recently, mixtures of this and common insulin). The adjustment between diet and insulin was carefully watched, the family being taught to carry out the daily control as well as the method of correcting the small maladjustments. In this manner the syndrome of Mauriac was cured in a few months, with complete disappearance of the symptoms, restoration of growth, and normalization of the blood chemistry. This shows that intense and prolonged treatment with insulin is not the cause of the appearance of the syndrome and that, on the contrary, a well adjusted treatment with sufficient insulin to reduce the levels of blood glucose before feeding to normal or almost normal values makes the syndrome disappear completely. The syndrome is never seen in children who are correctly treated from the beginning of the disease.

The author insists especially on a complete treatment of the diabetic child, medical, environmental, and social. She advises one: to make sure of a normal state of nutrition and development, from the somatic and functional points of view; to maintain normal blood sugar before eating, of 1.50 gm. or less per 100 cc.; to prevent sugar from appearing in the urine or to permit only small and rare quantities of it; to maintain cholesterol-blood levels lower than 2 gm. per 100 cc., and to avoid the inferiority complexes. In conclusion, the main characteristics of the nine cases are reported.

FROM THE AUTHOR'S SUMMARY.

ADRENOGENITAL SYNDROME. S. J. DE JONG, Maandschr. kindergeneesk **17**:155, 1949.

Two infants suffered from frequent vomiting and dehydration, caused by a lack of the "water and salt" hormone of the adrenal cortex. Both children had an increased excretion of 17-ketosteroids in the urine, especially of the fractions formed in the adrenal cortex.

The author suspects the presence of a tumor of the androgenic zone of the adrenal cortex, causing the virilization present in both infants (the first, a boy, had macrogenitosomia praecox; the other, a girl, had pseudohermaphroditism). This tumor has obliterated the other zones, resulting in the adrenocortical insufficiency.

Administration of desoxycorticosterone acetate saved the patients' lives by decreasing the lack of the "water and salt" hormone. FROM THE AUTHOR'S SUMMARY.

Diseases of Muscles, Bones, Joints, and Lymph Nodes

ARTICULAR DISORDERS IN CHILDHOOD. JULIUS B. RICHMOND and BESIE L. LENDRUM, Illinois M. J. **101**:295 (June) 1952.

This is a report of the results of cooperative studies in the Departments of Orthodontia and Pediatrics at the University of Illinois. The studies concerned problems of early diagnosis of rheumatoid arthritis in children.

In many children with rheumatoid arthritis the disease is undiagnosed unless they go on to the severer form of the disease. Articular manifestations in childhood often are monoarticular at the onset. The employment of blood cultures, agglutination, and skin tests is important in differential diagnosis. Differentiation from rheumatic fever may be troublesome. Palindromic rheumatism may be confused with rheumatoid arthritis.

If specific joint involvement is severe, interference with epiphyseal growth may develop. One area in which such growth disturbances occur most commonly has been the temporomandibular area with the resultant slowing of mandibular growth. The authors observed a relatively high incidence of this mandibular underdevelopment in children with rheumatoid arthritis.

The disease tends to be self-limited—40% of the patients recover completely. Therapy is largely nonspecific and supportive. Rigid immobilization of joints has not been helpful. Intensive vitamin D therapy no longer has any place. Corticotropin and cortisone are of value for experimental study, but for the present are of little practical value in the management of these patients. All of the patients under observation manifested psychological disturbances.

BARBOUR, Peoria, Ill.

DEPOSITION OF FLUORINE IN THE BONES AND TEETH OF THE GROWING RAT. I. ZIPKIN and F. J. MCCLURE, J. Nutrition **47**:611 (Aug. 11) 1952.

The results of Zipkin and McClure agree with previous evidence that the animal body accumulates fluorine at a comparatively rapid rate during growth, but reaches a relatively constant level of skeletal fluorine at maturity. This is maintained throughout the remaining years of life so long as the fluorine intake remains stabilized. FREDEEN, Kansas City, Mo.

ENGELMANN'S DISEASE (PROGRESSIVE DIAPHYSEAL DYSPLASIA). LEROY S. LAVINE and MORRIS T. KOVEN, J. Pediat. **40**:235 (Feb.) 1952.

Engelmann's disease, a rare syndrome first described in 1929, is marked by fusiform expansion and hyperostosis of the diaphyses of the long bones together with muscular weakness and nutritional failure. Roentgen studies show progressive, symmetrical, endosteal, and periosteal new-bone formation in the diaphysis of long bones. Under the microscope, the lesion is that of solid osteopetrosis. It is interesting that similar skeletal lesions have been produced in chicks by intravenous injection of human myeloid leukemic material.

Lavine and Koven report the syndrome in a 12-year-old boy. MACDONALD, Pittsburgh.

INFANTILE CORTICAL HYPEROSTOSIS. LOUIS G. BUSH and ORVILLE E. MERRELL, J. Pediat. **40**:330 (March) 1952.

A 4-month-old infant with cortical hyperostosis was given corticotropin, 10 mg. every 6 hours for 24 doses; 5 mg. every 6 hours for 24 doses; 5 mg. every 12 hours for 4 doses, and then 5 mg. every 24 hours for 3 doses. There was rapid clinical improvement. There were no x-ray changes in the bone lesions one month after treatment, but x-rays were normal four months after treatment.

MACDONALD, Pittsburgh.

VALUE OF ELECTROCARDIOGRAPHY IN PEDIATRICS. J. LUDER, Great Ormond St. J. (No. 3) 1 (June) 1952.

The history of the new technique of electromyography is discussed, and the application of this investigative method to clinical practice is described. As is pointed out by illustrations of various cases, the method may be useful in prognostic and diagnostic decisions.

In the case of trauma, the electromyographic curve will show whether complete transection of a valve has occurred or whether any continuity exists, thus indicating the need or lack of advisability for surgery. In facial palsy, the electromyogram will help to localize the particular areas affected and enables more accurate electrical and physical treatment to be given. By serial studies recovery can be followed and degree of eventual recovery can be estimated. When no further recovery can be expected, as shown by successive electromyograms, operations may be indicated. It is pointed out that physical examination alone will not show whether a good result can be expected.

The technique has been found useful in early or differential diagnosis of the amyotonias, myopathies, and muscular weakness or hypotonia in the early years of life. The author believes that the usefulness of electromyography will become more apparent with further clinical trial.

McGUIGAN, Evanston, Ill.

PES CALCANEO-CAVO-VALGUS. M. F. PILCHER, Great Ormond St. J. (No. 3) 36 (June) 1952.

Fourteen cases of paralytic pes calcaneocavovalgus treated surgically are reviewed thirteen to twenty years postoperatively. It is found that results generally are better functionally than anatomically. The authors feel that much of the deformity can be prevented by proper early tendon transplant and point out that such prevention gives far better results than late corrective operation.

McGUIGAN, Evanston, Ill.

MYOSITIS OSSIFICANS PROGRESSIVA TREATED WITH ACTH AND CORTISONE. P. R. EVANS, Great Ormond St. J. (No. 3) 56 (June) 1952.

Results of treatment with corticotropin (ACTH) and cortisone suggest that, while only palliative, these drugs may be used in relapses to prevent further calcifications.

McGUIGAN, Evanston, Ill.

BIRTH FRACTURE INVOLVING THE RIGHT TEMPOROMANDIBULAR ARTICULATIONS. A. M. TURNER, Great Ormond St. J. (No. 3) 68 (June) 1952.

Ankylosis of the right temporomandibular joint due to birth trauma resulted in facial asymmetry and severe trismus. Treatment was by arthrotomy, followed by exercises to develop greater range of movement.

McGUIGAN, Evanston, Ill.

SEVERE CASE OF GLANDULAR FEVER WITH ARTHRITIS. H. G. RISCHBIETH, Great Ormond St. J. (No. 3) 72 (June) 1952.

In a young child with many of the classical physical signs of glandular fever severe polyarthritis also developed. Recovery was not influenced by any form of drug therapy other than salicylates.

McGUIGAN, Evanston, Ill.

POLYOSTOTIC FIBROUS DYSPLASIA. J. R. D. WEBB, Proc. Roy. Soc. Med. **42**:357 (July) 1949.

The patient, a boy aged 5 years, was seen in June, 1948, with a history of vague ache in the right hip and of difficulty in walking. The child was quite well otherwise. Five brothers and sisters were all normal. On examination a bony swelling was noted in the region of the left frontal bone with orbit displaced downward, and the lower end of the left ulna was absent. While in the hospital the boy had a mild pyrexia. The chest was normal. A report on investigations, a roentgenological report on the bones, a report on a biopsy from the frontal bone and a full summary of the condition by Lichtenstein are included. Pictures of the following x-rays are given: one of the face shows rarefaction of the frontal bone; an x-ray of the humerus shows a trabeculated appearance; an x-ray of the upper end of the right femur shows rarefaction suggestive of cystic disease, and an x-ray of the ulna shows rarefaction and also absence of the lower end.

WILLIAMSON, New Orleans.

OLLIER'S DISEASE (DYSCHONDROPLASIA). C. T. POTTER, Proc. Roy. Soc. Med. **42**:359 (July) 1949.

In a boy aged 12 years the author reports a severe case of Ollier's disease, demonstrating extensive bilateral defects with predominant basic unilateral pattern, and roentgenological evolution of lesions. The first manifestation was noticed at 3 years when swelling of the terminal phalanx of a little finger was noticed. The patient was first seen at 6 years of age with a three weeks' history of a limp. The history, physical examinations, investigations, x-ray studies, and progress of the case are given fully in the report.

WILLIAMSON, New Orleans.

A VERY LARGE CONGENITAL LYMPHANGIOMA, TREATED SURGICALLY. G. POSTERARO, Lattante **20**:559 (Sept.) 1949.

A congenital lymphangioma the size of a watermelon, over the left side of the thorax, abdomen, and back of an 11-week-old infant, was successfully treated surgically after x-ray therapy had failed to reduce the size of the tumor.

HIGGINS, Boston.

RESULTS OF TREATMENT OF MULTIPLE CONGENITAL STIFFNESS OF JOINTS. U. MULLER, Schweiz. med. Wchnschr. **80**:1269 (Nov. 25) 1950.

Among orthopedic material of 40 years eight cases of arthrogryposis multiplex congenita were seen with periods of observation of 10 to 38 years. In addition, three children below 4 years of age have been under treatment for a short time. No other malformations were seen in any of the families of the patients. The result of treatment is limited by the want of muscle function. A certain improvement of motility can occur spontaneously. Treatment may be successful in that the patient can earn a living. But often all that can be accomplished is that the patient can attend to his daily needs without assistance by others. The treatment proceeds step by step. The first objective is to make it possible for the children to walk. Most important is continuous exercise of the muscles. Of the eight patients, four can walk well or satisfactorily without aids. The other four need apparatus or crutches. All can eat without help, although two or three with only one hand. Two of the three adults earn their living; the third can work a little. Five patients 10 to 15 years of age go to school.

AMBERG, Rochester, Minn.

A CONTRIBUTION TO THE KNOWLEDGE OF MYOTONIA CONGENITA (THOMSEN). FRIEDRICH KARL FRIEDERISCICK, Arch. Kinderh. **137**:41, 1949.

The diagnosis of myotonia was made in four siblings, one 12-year-old girl and her three brothers, 10, 14, and 18 years of age. The family tree of three generations reveals that the father of the children, one uncle, brother of the father, and two daughters of this uncle were suffering from the myotonic conditions. The grandfather was healthy, but the grandmother and a sister of hers were known to have had myotonia. Thus, out of 13 persons in the family listed, 10 had myotonia, and only 3 were free of symptoms. The heredity is dominant; no predilection of the male sex was found in this family.

In all patients the myotonia was recognized from the age of three years to puberty. The intensity of the symptoms was influenced by the surrounding temperature; heat acted favorably, and cold unfavorably. In the two milder cases the myotonic symptoms nearly disappeared in warm or high surrounding temperatures. Therefore, these two patients were classified as having an "intermittent type of myotonia congenita."

ARON, Chicago.

Diseases of Eye

OPHTHALMOLOGICAL SERIAL EXAMINATIONS OF CHILDREN. J. BARTH and F. HEFEL, Schweiz. med. Wchnschr. **82**:1165 (Nov. 8) 1952.

Eye examinations done on 765 schol children determined amblyopia (weak vision without organic lesions or with organic lesions where the diminution of vision was disproportional to the lesion), strabismus convergens and divergens, insufficient convergence, exophoria in need of treatment, and nystagmus. The simple tests necessary are described. Thirty-eight children needed refraction and 65 needed treatment in schools for vision.

AMBERG, Rochester, Minn.

Cutaneous Diseases; Allergy

POLIOSIS. ROBERT LOUIS GILMAN and HENRY H. PERLMAN, J. Pediat. **40**:101 (Jan.) 1952.

The term "poliosis" is derived from the Greek root meaning "gray." It indicates areas of localized depigmentation of the hair, in contrast to the term "canities," which is used to denote graying or whitening of the hair of the entire scalp. This case, in a 10-year-old white boy, started suddenly. There are two distinct patches of gray hair now in areas where, at first, only a few depigmented hairs were noticed. He has been given calcium pantothenate. No improvement has been observed.

MACDONALD, Pittsburgh.

PITYRIASIS ROSEA IN CHILDREN. HENRY H. PERLMAN and IRWIN I. LUBOWE, J. Pediat. **40**:109 (Jan.) 1952.

Perlman and Lubowe present a comprehensive review of pityriasis rosea, a not uncommon disorder in children. Lesions may be macular or papular, or maculopapular, or, rarely, vesicopapular. The authors have detailed the disease from the "herald patch" through the variety of symptoms to the pleasant fact that, no matter what the therapy is, most cases clear in four to six weeks.

MACDONALD, Pittsburgh.

SUBACUTE MERCURY POISONING (ACRODYNIA) CAUSED BY PROTIODIDE OF MERCURY. ALFRED E. FISCHER and HORACE L. HODES, J. Pediat. **40**:143 (Feb.) 1952.

Fischer and Hodes report a boy aged 3 years 6 months with symptoms of acrodynia. He had received protiodide of mercury for the treatment of warts, but mercury was found in the urine only after the administration of dimercaprol. The authors suggest that dimercaprol may have diagnostic as well as therapeutic value.

MACDONALD, Pittsburgh.

ACRODYNIA TREATED WITH PRISCOLINE.® ANDREW E. GILLESPIE, Canad. M. A. J. **67**:418 (Nov.) 1952.

A case of severe acrodynia due to mercury is reported and treatment with dimercaprol failed to give relief. Institution of benzazoline (priscoline®) therapy resulted in immediate and spectacular improvement. It was necessary to maintain treatment for two months. It is considered that the distressing symptoms were due to dysfunction of the sympathetic nervous system and that the persistent use of benzazoline blocked these distressing sympathomimetic effects throughout the course of the disease.

MCGUIGAN, Evanston, Ill.

ELECTROENCEPHALITIC STUDY OF ALLERGIC CHILDREN. LINDA GROSSI-BIANCHI, Minerva pediat. 4:118 (Feb. 29) 1952.

Electroencephalograms are reproduced of four children with asthma. One of the tracings shows waves of the type found in psychomotor epilepsy; two others show occipital dysrhythmia. As the neurovegetative centers play a role in the etiopathogenesis of allergic conditions, it is suggested that electroencephalograms of allergic children may prove useful in detecting neurovegetative disorders and in anticipating eventual psychosomatic reactions by the proper treatment of the patient.

HIGGINS, Boston.

ACRODYNIA IN AN INFANT AFTER THE APPLICATION OF A MERCURIAL OINTMENT. EUCLIDES PELUFFO, JORGE NORDMANN, and PEDRO A. RIVERO, Arch. pediat. Uruguay 23:691 (Oct.) 1952.

A child 8½ months old, with eczema, after a local treatment with an ointment of yellow mercury oxide, presented a long process which lasted from October, 1951, till February, 1952, with a predominance of the clinical symptoms of acrodynia. There were psychological changes (change of character, sadness, drowsiness), photophobia, erythema with desquamation of the skin of the hands, greater muscular hypotonicity and increased blood pressure; there were also prolonged fever, serious malnutrition and important trophic disorders of the skin. There were also very significant respiratory disorders and anasarca following serous changes in protein metabolism (decrease in blood proteins). Mercury was found in the urine (90 γ per liter). He was treated with dimercaprol, although insufficiently, improving only partially and temporarily. Later he was treated with corticotropin. By the influence of this, or because the disease came to the end of its course, the patient recovered.

FROM THE AUTHORS' SUMMARY.

ETIOLOGY AND THERAPY OF SEBORRHEIC DERMATITIS. HEINZ LEMKE, Monatsschr. Kinderh. 98:350 (Aug.) 1950.

Seborrheic dermatitis in infants is due to lack of several constituents of the vitamin B complex. Synergism of biotin with the entire vitamin B complex has been shown; biotin has been proved to be essential and its combination with vitamin B_6 to be important. Nicotinic and pantothenic acids do not seem to be essential. Biotin deficiency in man can be produced by (1) qualitative and quantitative undernutrition and (2) intake of raw egg albumin. Therapy for seborrheic dermatitis in breast-fed infants consists of (1) ingestion of at least 1,500 gm. of cooked or roasted liver or 1,000 gm. of kidney by the mother, (2) three injections of 5 mg. biotin and 1 ampule of vitamin B complex, two days apart, into either mother or infant, and (3) local treatment of the skin. In artificially fed babies the diet should be casein-rich and low in fat (buttermilk); skin care should be given and injections of biotin and vitamin B complex should be carried out as for the breast-fed infant.

HARTENSTEIN, Madison, Wis.

OSLER'S DISEASE. JOACHIM WOLFF, Monatsschr. Kinderh. 98:431 (Oct.) 1950.

All four children of a family are reported to show the features of Osler's disease (telangiectasia hereditaria haemorrhagica). They had frequent epistaxis and showed telangiectasia of cheeks and lips. The family history revealed five other members with the characteristics of this disease. Prolonged administration of rutin should be tried therapeutically.

HARTENSTEIN, Madison, Wis.

STUDIES OF ENDOGENOUS HISTAMINE IN CLINICAL AND EXPERIMENTAL HYPOXEMIA. WILHELM GRAF, Acta pædiat. 38:180, 1949.

In congenital heart disease with cyanosis (tetralogy of Fallot, Eisenmenger complex) high blood-histamine values occur, although they do not give rise to mean values significantly different from those of healthy acyanotic children of the same age. Out of 12 cases of morbus caeruleus 2 yielded values over 3 σ higher than the mean value of the control group. These high values displayed no correlation with the degree of cyanosis or polycythemia.

The blood histamine level of normal children was identical with that of healthy adults. The means of the groups were 0.091 ± 0.21 (n equals 12) and 0.095 ± 0.023 (n equals 10) γ histamine HCl per cubic centimeter of whole blood, respectively.

Ten healthy adults given a 15-minute cardiological hypoxia test with 7 to 9% oxygen displayed no significant rise in the blood histamine at the end of the test, although two subjects showed values over 3 σ higher than the mean value before the test.

Mice suffocated slowly to death showed a histamine content per whole animal which, calculated per gram of body weight, was three times as large as that of normal animals.

Small amounts of histamine were demonstrated when trichloroacetic acid extraction was performed on 10 mg. of *d*-histidine and on 10 mg. of a compound obtained by coupling histamine with diazotized benzidine. Such errors are, however, not considered serious when the method in question is used for purposes of comparison.

FROM THE AUTHOR'S SUMMARY.

Miscellaneous

INTOXICATION WITH VERATRUM VIRIDE. ARTHUR J. NEWMAN, J. Pediat. **40**:233 (Feb.) 1952.

Veratrum viride, no new drug, is again becoming popular in the treatment of hypertension. The case reported here is of a 23-month-old infant who accidentally swallowed an unknown number of 1 mg. veratrum-viride tablets. Symptoms, rapidly progressive, were hypothermia, fall in blood pressure, sweating, nausea, vomiting, diarrhea, headache, and cardiovascular collapse. In this case, atropine was an effective antidote. Other antidotes are ephedrine and/or morphine.

MACDONALD, Pittsburgh.

ANTIBIOTICS IN CHILDHOOD. E. ROSSI, Helvet. paediat. acta **7**:397 (Nov.) 1952.

With the present state of antibiotic therapy, there is an ever-increasing need for a specifically aimed therapy. The introduction of resistance tests, mainly through the Bondi method, has greatly simplified this problem. The antibiotics act primarily in a bacteriostatic sense. A number of authors consider a simultaneous bactericidal action, especially with penicillin and streptomycin, as possible.

The increasingly observed resistance of the pathogens can arise through the proliferation of pathogens which were already resistant at the onset of therapy, or can occur during therapy because of as yet unknown factors. From this it is to be concluded that antibiotics are to be administered only upon strict indication.

The action of streptomycin and penicillin is synergic. Aureomycin, oxytetracycline ("terramycin"), and chloramphenicol are active by addition to their effects. With germs which are sensitive to medicaments of the first group, the combination with medicaments of the second group produces an antagonistic action; with sensitive germs, a synergic action.

Important in all antibiotic therapy, especially penicillin, is a sufficient dosage. A low dose often leads to resistance of the pathogens and sensitivity reactions in the patient. It is, however, to be remarked that too high an initial dose can lead to a worsening of the symptoms and is especially to be avoided in cases of congenital syphilis.

Side reactions occur mainly in the sense of allergic manifestations. The orally administered tablets and the nose drops do more harm than good in this respect. The inhibitory action of penicillin and chloramphenicol on the bone marrow also should be mentioned. Oxytetracycline can lead to severe states of dehydration as the result of sudden intestinal disturbances. Of importance today are also the fungous infections, which occur as a result of the destruction of the antagonistic-physiological flora in the intestinal tract and in the respiratory passages and are treated with paraben, according to recent experiences. Dihydrostreptomycin is to be eliminated because of the severe acoustic damage, and the regular streptomycin is to be used instead.

Penicillin remains the antibiotic of choice in Streptococcus, Staphylococcus, Meningococcus, and Pneumococcus infections. It is especially indicated in scarlet fever. Streptomycin is used today almost exclusively in the treatment of tuberculosis. Chloramphenicol is especially indicated in Salmonella and other intestinal infections, as well as in cases of Pfeiffer meningitis.

Aureomycin is used especially in infections caused by viruses of large molecular size, in Rickettsial diseases (Q fever for example) as well as in malignant infant enteritis caused by pathogenic coli. Oxytetracycline is used mostly in pyuria.

Aureomycin acts as a growth factor, and has already been applied as such in veterinary medicine with good results.

Thorough reflection, limited indications, specifically directed choice of antibiotics and optimal dosage are fundamental in antibiotic therapy.

FROM THE AUTHOR'S SUMMARY.

COMPARATIVE INVESTIGATIONS WITH REGARD TO THE THERAPEUTIC EFFECT OF CORTICOTROPIN OR SODIUM CITRATE IN EXPERIMENTAL LEAD INTOXICATION. S. MOESCHLIN and L. SCHECHTERMAN, Schweiz. med. Wchnschr. **82**:1164 (Nov. 8) 1952.

Guinea pigs received lead nitrate intraperitoneally. Treatment with dimercaprol was ineffective, perhaps even injurious. Sodium citrate by gavage proved definitely beneficial; indeed, some animals survived and recovered completely. For treatment of patients, immediate administration of sodium citrate in solution by mouth is recommended, together with other symptomatic treatment of colic, obstipation, and spasms of the colon.

AMBERG, Rochester, Minn.

A NEW METHOD FOR INTRAVENOUS INFUSION OF FLUIDS IN CHILDREN, BY MEANS OF A PLASTIC CANNULA. G. CERNUSCHI and M. SANVILLI, Clin. pediat. **31**:571 (Oct.) 1949.

This article outlines the methods in use in American clinics, as described by Alexander, Small, and Campbell (*Ann. Surg.* **127**:1212 [June] 1948) and by Arnold and Arnold (*J. Pediat.* **32**:113 [Feb.] 1948).

HIGGINS, Boston.

THE ABSORPTION, EXCRETION, AND DOSAGE OF SODIUM PARAAMINOSALICYLATE IN CHILDREN. C. TORRICELLI and S. CAPPANERA, Pediatria **57**:650 (Sept.) 1949.

The pharmacology and the clinical use of sodium paraaminosalicylate are discussed. A daily dose of 0.3 to 0.4 gm. per kilogram of body weight for infants and small children, and of 0.3 gm. for older children, is recommended. Methods of chemical analysis of paraaminosalicylate in urine and blood are described. Tables are given on data as to the blood and urine concentrations of paraaminosalicylate in children after its administration by the oral, the intramuscular, the subcutaneous, and the rectal route.

HIGGINS, Boston.

CONCENTRATIONS OF POLYSULFONAMIDES IN THE URINE AND BLOOD OF CHILDREN. M. RAVERA, Minerva pediat. **1**:337 (Sept.) 1949.

Polysulfonamide therapy consists of the giving of a mixture of sulfonamide drugs instead of a single drug. Polysulfonamide medication led to a higher blood level of sulfonamide drug, which persisted longer and was relatively less toxic than when a single sulfonamide drug was given in the same dosage.

HIGGINS, Boston.

THERAPEUTIC USE OF PROCAINE IN PEDIATRICS. E. GRASSO, Minerva pediat. **4**:111 (Feb. 29) 1952.

After discussing the chemistry, the pharmacology, the dosage, and the possible toxicity of procaine as intravenous medication, the author outlines the uses of the drug in pediatric practice. Procaine administered intravenously will relieve the toxic symptoms in various infections, such as dysentery and bronchopneumonia. This beneficial effect is by the effect on the sympathetic nervous system; in other words, the symptoms are affected rather than the actual lethal toxic action on the cells.

Procaine administered intravenously has also been found to relieve patients with pertussis by cutting down the frequency and severity of the coughing paroxysms. Relief of the visual disturbances (amaurosis) frequently encountered in tuberculous meningitis is reported following intravenous administration of procaine. Many of the severer symptoms of transfusion reactions.

including hyperthermia, in sick children, have been relieved by this drug. In acute glomerular nephritis, intravenous administration of procaine has been reported to induce diuresis and to lower the blood pressure.

One hundred and nine references are listed, mostly from the foreign literature in this extensive review.

HIGGINS, Boston.

ORAL PENICILLIN IN INFANTS: STUDY OF THE BLOOD LEVELS. CARMEN AGUILO, JULIO MENEGHELLA and RENE PELLICER, Rev. chilena de pediat. **20**:320 (Aug.) 1949.

The penicillin-blood levels obtained in three samples of serum from malnourished infants after the oral administration of from 4,000 to 7,000 units (per kilogram) of an acid and alkaline solution of penicillin were compared with the levels obtained after the intramuscular injection of 1,000 to 2,700 units per kilogram of body weight.

The results were analyzed from the following points of view: absorption, duration of blood levels, bacteriostatic concentrations, age, dosage, importance of the alkalines, comparison with the results obtained by foreign authors, and superiority of the intramuscular over the digestive route.

After oral administration of penicillin the antibiotic can be detected in the blood in 90% of the cases. The concentrations and the maintenance of these levels vary in different individuals. In 64% of the cases bacteriostatic concentration can be found for at least 90 minutes. The authors did not find age to be an important factor. They did not find that adding aluminium hydroxide in a colloidal state had any advantages. This route of administration for penicillin treatment should be further investigated, especially in infants.

FROM THE AUTHORS' SUMMARY.

TOXICOLOGY OF AMINOPYRINE. R. KOCH and A. HÜBNER, Med. Klin. **44**:1571 (Dec. 9) 1949.

A boy 8 years of age received an intravenous injection of 10 ml. of a 20% aminopyrine solution intravenously within four minutes. Severe toxic clonic convulsions started immediately. Intravenous injection of a barbiturate controlled the condition successfully. A few cases of death after oral administration in children are cited. The authors limited the intravenous administration of aminopyrine to rheumatic diseases of children not below 5 to 6 years old with a maximal dosage of 0.8 gm.

AMBERG, Rochester, Minn.

ACUTE IRON "POISONING." NILS LINDQUIST, Acta pædiat. **38**:447, 1949.

Ferrous chloride, which has been considered a completely harmless iron preparation, may, like large doses of ferrous sulfate, prove very dangerous, at least to children. On several occasions there have been references in English literature to "poisoning" with the latter. A case is reported here of ferrous chloride poisoning in a 2½-year-old girl who took a single dose of about 1,500 mg. $FeCl_2$ with resultant necrosis of the mucous membrane of the stomach, hematemesis, blood in the stools, and a condition of collapse. Even more innocuous medicines should be kept out of the reach of children.

FROM THE AUTHOR'S SUMMARY.

News and Comment

ANNOUNCEMENTS

Course in Rheumatic Heart Disease.—The Cook County Graduate School of Medicine announces a two-week intensive course on the diagnosis and treatment of congenital and of rheumatic heart disease in infants and children, presented by Benjamin M. Gasul, M.D., and Egbert H. Fell, M.D., and associates, from May 18 to May 30, 1953. For a circular giving full information, write to Registrar, Cook County Graduate School of Medicine, 707 South Wood St., Chicago.

Postgraduate Course in Pediatrics.—A two weeks' postgraduate course in general pediatrics will be given at Burnham Memorial Hospital for Children, Massachusetts General Hospital, Boston, under the auspices of the Harvard Medical School and direction of Dr. Allan M. Butler, Professor of Pediatrics, June 8 through June 20, 1953. Clinics providing a broad review of pediatrics, emphasizing the new advances, will be given daily from 9:00 a. m. to 4:30 p. m. by selected authorities from the Pediatric and Specialty Services of the Massachusetts General Hospital and the Children's Medical Center, Boston.

For further information write Assistant Dean, Courses for Graduates, Harvard Medical School, 25 Shattuck St., Boston.

Fellowship in Pediatric Cardiology.—A fellowship in pediatric cardiology at the University of Utah is being offered for one year starting July 1, 1953. Both clinical and laboratory training in congenital and rheumatic heart disease will be given, and will include cardiac catheterization and angiocardiography.

Applicants will be considered on the basis of former training and military status. The annual stipend will be $3,000 for an unmarried fellow and $3,600 for a married fellow. Applicants should write to Dr. L. George Veasy, Department of Pediatrics, University of Utah College of Medicine, Salt Lake General Hospital, Salt Lake City.

Books

Children's Radiographic Technic. By Forrest E. Shurtleff, R.T. Price, $3.75. Pp. 80, with 32 figures. Lea & Febiger, 600 S. Washington Sq., Philadelphia 6, 1951.

This book is concerned with roentgenographic techniques peculiar to children. It is divided into four chapters, which are (1) Equipment and Accessories; (2) Children's Radiographic Positions; (3) Special Radiographic Procedures, and (4) Children's Technic.

Equipment for roentgenography of children is the same as that used for adults. To lessen motion, the shortest possible exposure time is used. Accessories, consisting of a cotton-webbing strap, nonstationary head holders, balsa-wood boards, folded sheets, and an open-end cassette holder, are simple and easily obtained, and their use as an aid to help prevent motion is discussed.

The various roentgenographic positions are described, and practical methods of obtaining cooperation are suggested. Sedation can be used when necessary. Frequently children are held in the required position by attendants. Certain views for better demonstration of the manifestations of generalized diseases affecting the skeleton are suggested.

Special procedures consist of examination of the gastrointestinal tract, bronchography, intravenous urography, tracheograms, rhinograms, encephalograms, and other similar procedures.

The fourth chapter consists of tables with a complete roentgenographic technique developed for age groups from birth to 12 years of age.

This volume provides an excellent technical reference book for any department which has to deal with the roentgenography of children.

Review of Physiological Chemistry. By Harold A. Harper. Price, $3.50. Pp. 260. University Medical Publishers, P. O. Box 761, Palo Alto, Calif., 1951.

The author's preface indicates that this book was designed to present concisely the fundamentals of biochemistry with emphasis on accepted facts and concepts as a supplement to standard textbooks. Much of the material in the book is drawn from the author's lectures in basic science given in conjunction with the graduate training of physicians. It is hoped that the book will serve as a review for the physicians preparing for state and specialty boards. Twenty-one well-organized chapters cover the essentials of intermediary metabolism of food materials, vitamins, enzymes, hormones, mineral and water metabolism and electrolyte equilibrium in body fluids, renal and hepatic function, etc. There are excellent tables listing data on the composition of body fluids and tissues, dietary requirements and normal values for various functional tests. Many diagrams represent normal metabolic pathways of intermediary metabolism and disturbances thereof in disease. The book packs a wealth of factual information.

Color Atlas of Morphologic Hematology with a Guide to Clinical Interpretation. By Geneva A. Daland. Price $5. Pp. 74, with 12 tables and 9 illustrations (14 color plates). Harvard University Press, Cambridge 38, Mass., 1951.

The purpose of this book is to furnish a guide for reference in the study of films of peripheral blood stained with Wright's stain. This has been well accomplished by the authors. The first part of the book deals with the values and limitations of the blood film in diagnosis, preparation of blood films, and the general characteristics of formed elements of blood stained with Wright's stain, followed by a maturation description of the blood cells. This is followed by descriptive colored plates showing the various disorders of the blood.

The beauty of this book is its simplicity in that it is compiled from a purely clinical point of view. No attempt is made to discuss in detail the many theories regarding the origin of the cells, but emphasis is placed on the significance of the findings from a diagnostic, prognostic, and therapeutic point of view. The colored plates are excellently done and the subject well chosen. The entire book is well bound and printed and makes a valuable edition to the library of anyone interested in the morphology of the blood.

Physical Diagnosis. By Raymond W. Brust, M.D., F.A.C.P. Price, $4.50. Pp. 300, with 71 illustrations. Appleton-Century-Crofts, Inc., 35 W. 32d St., New York 1, 1951.

This volume is a concise, well-organized, well-illustrated, and thorough text on physical diagnosis admirably suited to the needs of the medical student or beginner in the art of recognizing the physical signs of disease. It is organized by anatomical symptoms and diseased states. The author wisely does not digress to discuss at length the etiology and symptoms of various diseases. When necessary he makes a concise statement of the accompanying symptoms. He states clearly the physical bases and meaning of useful physical signs and men's names associated with them, pointing out many things valuable to the beginner.

The author discusses the physics of sound and resonance as related to the chest and head. He describes fully both normal and abnormal signs related to the heart and lungs from the standpoint of adult examination. There are numerous omissions of findings peculiar to infancy. For example, the section on cardiovascular phenomena makes no mention of the absence in early infancy of the characteristic murmur of patent ductus arteriosus. A chapter on cranial nerves is a good résumé of the physical signs referable to them but does not mention retrolental fibroplasia.

To summarize, this text fulfills the need for a thorough concise treatise on physical diagnosis for the beginner, but it is not a pediatric text.

Disease In Infancy and Childhood. By Richard W. B. Ellis. Price, $8. Pp. 695, with 296 illustrations. Williams & Wilkins Company, Mount Royal and Guilford Aves., Baltimore 2, 1951.

This new pediatric text is unusual in these times in that it is the work of a single author, who is professor of child life and health at the University of Edinburgh.

The text is divided into 22 chapters, which cover adequately, for the most part, the field of pediatrics. The initial chapter deals with history-taking and examinations. In this section an interesting inclusion is a scheme for a psychological history for use in behavior problems. While it is designed for the social worker in large part, it is a valuable outline for the pediatrician. In this chapter also is included very brief mention of growth and development, far too brief for our present preoccupation with this important phase of child health.

Two following chapters are concerned with social and environmental factors in disease and constitutional factors in disease. Both of these are excellent and are not generally found in other texts.

Following these introductory chapters the author discusses the field of pediatrics in general in an orderly sequence of systems, beginning with the newborn and congenital malformations and proceeding through the various systems. Included are chapters on disorders of nutrition and digestion; disorders of storage and disorders of growth and development, which two chapters include diabetes and other metabolic disorders; neoplastic diseases; allergic disorders; rheumatic disorders; degenerative and miscellaneous disorders, and behavior disorders. There is no section on orthopedics, various orthopedic disorders being considered in different sections.

Within its limitations this is an excellent up-to-date text. The format and illustrations are very good. In size the book is midway between a handbook and a text, and its usefulness is likely to be in proportion to its size. The unusual fact of a single author gives the book a continuity and homogeneity lacking in American texts, and few if any faults can be found with the subject matter included.

In view of the necessary brevity entailed in presenting the scope of pediatrics in a text this small, the author has done an admirable piece of work. The book is interesting reading and can be well recommended. Its course in future editions will be watched with interest.

Tim and His Hearing Aid. By Eleanor C. Ronnei, Max Porter and John Porter. Price, $1.75. Pp. 44, with 44 illustrations. Dodd, Mead & Company, Inc., 432 4th Ave., New York 16, 1951.

This is a little book written for children who are hard of hearing, but it is also instructive for the parents and teachers, presenting the difficulties which may occur in getting a youngster to wear one of these aids. It is a good book.

Management of Celiac Disease. By Haas and Haas. Price, $5. Pp. 188. J. B. Lippincott Company, 227-231 S. 6th St., Philadelphia 5, 1951.

This little book is easily read. It contains a bibliography of 668 articles. The authors have had wide experience, having diagnosed 603 cases as celiac disease and having followed 370 of them for a long enough period to evaluate therapy with the "carbohydrate specific diet." The authors differentiate fibrocystic disease of the pancreas from celiac disease and include in the latter steatorrhea and carbohydrate intolerance.

General practitioners and pediatricians will find this book of value in the recognition and treatment of celiac disease.

Surgical Care: A Practical Physiologic Guide. By Robert Elman, M.D., F.A.C.S. Price, $8. Pp. 586, with 18 illustrations. Appleton-Century-Crofts, Inc., 35 W. 32d St., New York 1, 1951.

Unfortunately knowledge is not inherited. Every young surgeon must absorb an infinite number of facts about the entire field of surgery—so much to learn, so little time. This book is the answer to the surgeon's need for basic knowledge of the general care of surgical patients. The author has taken from his own experience and from the teachings of others the physiologic time-tested and accepted guides to good surgical care and has set them forth in a well-written, orderly, and readable book.

The historical references at the beginning of many of the chapters add spice and are a stimulant to those who are interested in the history of surgery.

The chapter on "Pyschogenic Factors in Surgery" should be required reading for every intern and resident, even for a few older surgeons. It emphasizes so calmly, yet firmly, that a patient is first, last, and always a human being, anxious, and fearful about the operation.

A pleasing insight into the character of the author is obtained from this paragraph on the need for cooperation from all personnel in the care of a surgical patient: "The best type of response in general will be forthcoming when the surgeon is thoughtful and considerate of the rights and feelings of the personnel under him or associated with him. It is true that some surgeons may deny this statement and maintain that a dictatorial attitude evokes the best cooperation. In most cases, however, experience has shown that the opposite view is correct, and that consideration in his dealings with staff personnel is the best way to gain the utmost in teamwork."

This book is especially valuable to the young surgeon and the busy surgeon who wish to learn the essentials of good surgical care but do not have time to search through the endless volumes of surgical literature.

A Pediatric Manual for Mothers. By Harry R. Litchfield, M.D., and Leon H. Dembo, M.D. Price, $2.50. Pp. 256. Grune & Stratton, Inc., 381 4th Ave., New York 16, 1951.

This is the newest book in the series directed to mothers. It is not an antepartum survey. The authors are well qualified to answer questions on the basis of 30 years' relationship to mothers.

The subjects are very contemporary, including such chapters as Dental Caries and the Fluorine Treatment; Surgical Treatment of Congenital Hearts; Allergy in Infants and Children; ACTH and Cortisone; The Rh Factor; The Wetzel Grids; Traveling with Baby; Baby Sitters, and Movies, Radio, Comic Books, and Television, as well as a wide range of more usual subjects. They cover physical, mental, and developmental problems.

The style is a question and answer format which makes it easy to refer to a given subject quickly. The index is extensive and the language is simple, avoiding medical "gobbledegook." The book is similar to Holt's "Care and Feeding of Children" and seems to cover as much in a more concise manner.

The answers given are in keeping with majority medical and psychological opinion.

Occasionally the authors are a bit dogmatic, but perhaps that is a virtue in writing for the lay reader. This "primer" can very well be recommended to mothers.

You and Your Heart. By Drs. H. M. Marvin, Irving S. Wright, Irvine H. Page, T. Duckett Jones and David D. Rutstein. Price, $3.00. Pp. 306, with 1 illustration. Random House, Inc., 457 Madison Ave., New York 22, 1950.

This concise, readable book of 306 pages including the index entitled "You and Your Heart" is written by a group of such eminent specialists as Dr. H. M. Marvin, President of the American Heart Association and Associate Clinical Professor of Medicine at Yale University; Dr. Irving S. Wright, President of the New York Heart Association and Professor of Clinical Medicine at Cornell University; Dr. Irvine H. Page, Director of Research of the Cleveland Clinic Foundation and Chairman of the Medical Advisory Board Counsel for High Blood Pressure Research of the American Heart Association; Dr. T. Duckett Jones, Medical Director of the Helen Hay Whitney Foundation, and Dr. David D. Rutstein, Professor of Preventive Medicine, Harvard University.

Amazingly, the book covers every common kind of heart and blood vessel difficulty. It is informative to anyone who is merely curious about the heart and the arteriovenous system. To me, a physician who left school 20 years ago and who has consistently read books and journals, it afforded new knowledge and understanding of various aspects of heart disease.

The book can be recommended by physicians to patients with coronary disease. It will certainly make the busy physician's work easier. It will make the patient far more aware of the nature of his problem and, I believe, definitely less fearful, because the book is written reassuringly and authoritatively. It answers questions such as what are congestive heart failure, arteriosclerosis and atherosclerosis, and it gives fairly specific advice about exercise, smoking, alcohol and work.

The book has a great deal of information for the parents of children with congenital heart disease. There is advice for people who worry about nonexistent heart trouble. There is information for those who wish to learn about Buerger's disease (thromboangiitis obliterans), Raynaud's disease and thrombophlebitis. The chapter on misapprehensions about heart disease will aid any patient with heart disease and clarify the common misunderstanding of many.

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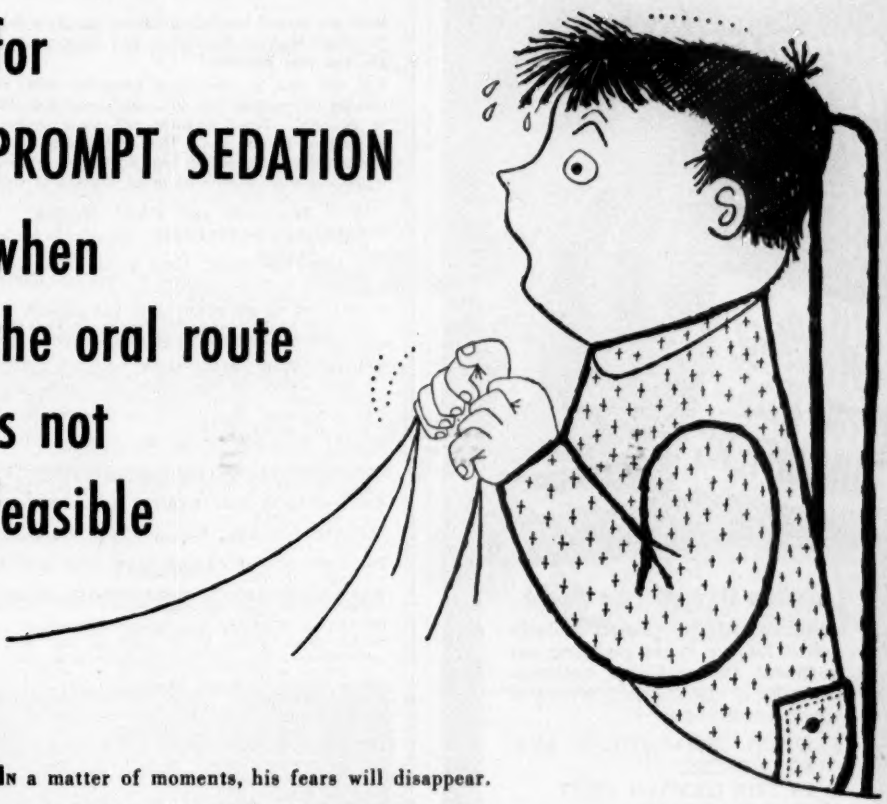

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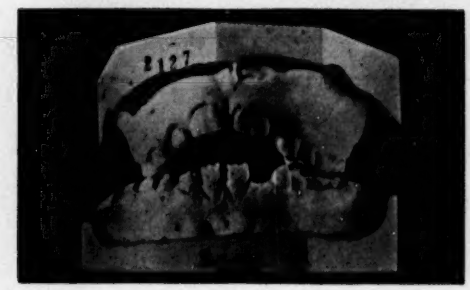

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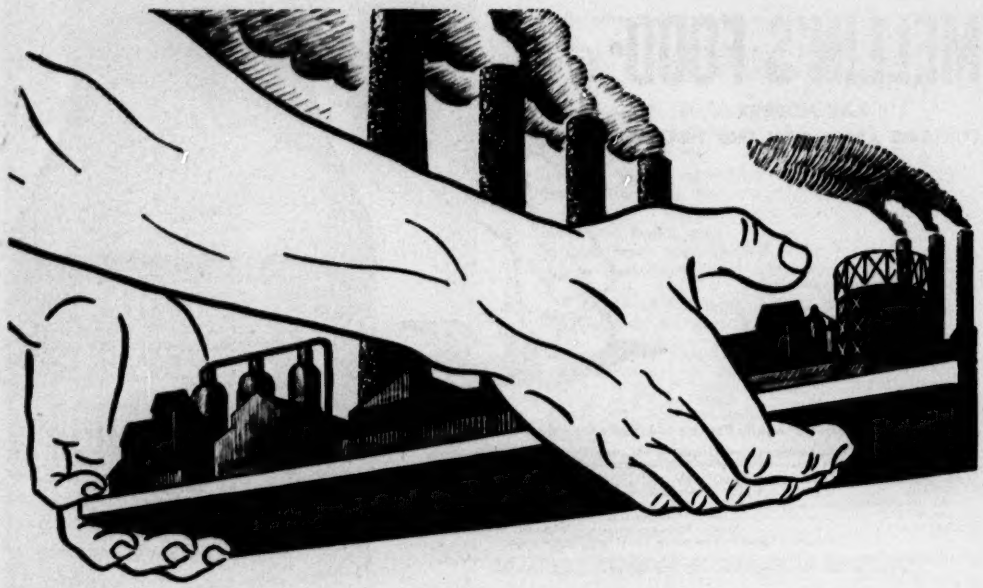

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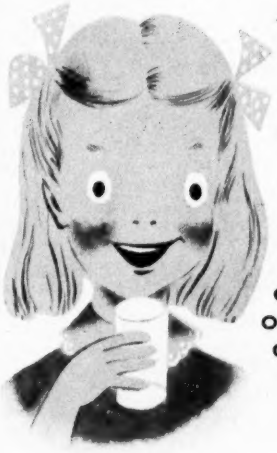

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